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THE TOXICOLOGY OF THE NEWER METALS

BY

LAWRENCE T. FAIRHALL

From the Industrial Hygiene Division, United States Public Health Service

In the following discussion of the toxicology of the ' newer ' metals, it is perhaps more accurate to refer to them as metals which have only recently become of industrial importance. Most of the metals mentioned have long been known, and in some instances have actually been used in quantity. However, many new types of use have developed, or unusual compounds have become important industrially and have aroused the interest of the industrial hygienist.

It should be realized that the metals and their compounds in general have distinctive characteristics, and that it is somewhat difficult to group their physiological action in the same way as is possible with great groups of organic compounds. For instance, the nitro or amino compounds, or the halogenated hydrocarbons, have many characteristics which permit some evaluation of the toxic qualities of the individual members of the group from what we know of the group in general. The behaviour of the metals and their compounds is far less predictable from this point of view. Characteristics which belong to the group as a whole may vanish with one particular compound, or vice versa. Thus, tin salts are quite harmless. Individuals daily consume quantities of tin that would be a matter of concern if tin were as toxic as many other metals generally regarded as harmless. Yet tin tetrahydride is stated to be one of the most toxic substances, being more poisonous than arsine. The following discussion, therefore, tries to evaluate the known facts about the physiological action of the metals and their compounds as separate entities. Very little is known of some of these substances, either because they have been of no great commercial importance, or because heretofore they have not displayed properties which are distinctive in a physiological sense.

Beryllium

A few articles have been added to the recent literature concerning the industrial hazards associated with the production of beryllium, but during the past three years no work has been reported on the physiological action of beryllium itself. Shilen, Galloway, and Mellor (1944) reported the health hazards incident to the extraction of beryllium. Kress and Crispell have reported cases of chemical pneumonitis in men working with fluorescent powder containing beryllium. Van Ordstrand, Hughes, De Nardi and Carmody have recently reported the dermatological and other effects which they have observed in the beryllium industry during the past four years, and which were reported under the heading of ' beryllium poisoning.' The fatal cases described in this report resulted from chemical pneumonitis. A similar chemical pneumonitis developed in an additional 33 workers at various occupations in these plants. While the hazard due to dust and fume that existed in the plants was undeniable, the term ' beryllium poisoning ' is a misnomer, since no toxic action can be directly attributed to the beryllium ion itself, and the beryllium in these cases merely acted as an adjuvant.

In connexion with the manufacture of fluorescent lamps, a number of cases designated as sarcoidosis were reported in 1943 in the Massachusetts area. Since beryllium oxide is one of the constituents of the fluorescent powder, and since very little was known concerning the toxicity of beryllium, this substance was suspected as the aetiological factor in the production of sarcoidosis.

The inhalation of beryllium sulphate dust was shown to be injurious to guinea-pigs, 67% mortality in one exposure, in experimental work reported by Hyslop *et al.* in 1943; but, when the more neutral potassium beryllium sulphate was substituted, the animals were able to tolerate much larger doses; there was no mortality in large doses daily for seven days. Later experimental work, using rabbits, has confirmed this earlier finding.

Beryllium sulphate is so extensively hydrolysed that hydrogen is evolved when zinc is placed in a solution of the salt. It is understandable that the inhalation of beryllium sulphate dust would prove strongly irritating, owing to the local production of sulphuric acid in contact with the delicate tissue of the lung substance. The production of a pneumonitis in the victim exposed to such irritating material is readily understood. Potassium beryllium sulphate, on the other hand, is soluble in cold water, is nearly neutral in reaction, and yields all the reactions characteristic of the beryllium ion. It is not bound as a complex, which might be expected to have different properties from other beryllium salt solutions. If beryllium were a protoplasmic poison, both beryllium sulphate and potassium beryllium sulphate should show similar effects. With reference to the ulceration produced by beryllium sulphate entering cracks in the skin—a condition which occasionally occurs with employees handling this material—it would be of interest to try the effects of beryllium salts of more inert acids, such as beryllium citrate or beryllium malate.

Cadmium

In spite of the increased attention which has been devoted to the hygienic significance of cadmium, cases of poisoning—usually resulting from unsuspected origin—appear with sufficient frequency to re-emphasize the fact that cadmium is a potentially dangerous substance. The extensive report of Spolyar and his associates on cases of cadmium poisoning resulting from flanging operations on cadmium-plated pipe gives a particularly clear insight into the type of danger resulting from exposure to cadmium oxide fumes. Five cases, including one death, are summarized. On the basis of the 59 cases reported in the literature, the mortality rate of industrial cadmium poisoning appears to be 15%.

As a result of cases of cadmium poisoning arising from the heating of cadmium-coated metal—with the attendant formation and volatilization of dangerous quantities of cadmium oxide—the labelling of cadmium-coated metals has been urged. While this measure is effective

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for large pieces, it is somewhat difficult to ensure that small objects so coated are labelled. A very convenient test that can be directly applied to the cadmium-coated surface depends upon the formation of a yellow cadmium sulphide stain on the metal surface.

Cases of cadmium poisoning from careless use of vessels as beverage containers continue to be reported. In 1944 Lufkin and Hodges reported twelve men as having been affected by artificial lemonade prepared in metal containers which were later shown to be cadmium-coated. The significant feature of cadmium poisoning by mouth is the rapidity of physiological response. The effects usually occur within 15 to 30 minutes after ingestion of food or drink containing toxic quantities of cadmium. This in itself should be suggestive in cases of so-called food poisoning. Occasionally, however, symptoms may be delayed as long as four or five hours.

Although cases of extensive cadmium poisoning have been reported from the use of cadmium-coated ice-cube containers in refrigerators, an unusual source of cadmium contamination was reported by Shiftner and Mahler in 1943 as having affected seven persons: cadmium plating of the refrigerating unit itself was responsible. Flakes of cadmium had dropped into the ice-cube compartment. A sample of ice cubes from the trays contained 769 parts per million of cadmium. A survey of apartment houses showed that 25% of the refrigerators tested had cadmium-coated parts.

Ross has recently cited a case of mass poisoning due to cadmium oxide fumes, in which 23 individuals were affected. Finely divided cadmium dust from a cadmium-recovery chamber became ignited owing to red-hot cigarette ash carelessly dropped by one of the workers. In a few minutes the cadmium dust became incandescent, emitting clouds of cadmium-oxide fumes. Although no fatality occurred, incapacity was somewhat serious in some of the cases, disability amounting in one case to as much as two months. Ross's tabulation of the symptoms displayed by the 23 victims shows that praecordial constriction, dyspnoea, gastric pain and prostration were the predominant effects.

Delayed action is often reported to be characteristic of exposure to cadmium-oxide fume. The effects usually appear only 3 or 4 hours after exposure. The present maximum allowable concentration of cadmium fume in the atmosphere of workrooms is one milligramme per ten cubic metres of air (American Standards Association, 1941).

Cobalt

Cobalt has occupied rather a minor position in commerce; but, as new and surprising uses have developed, it has become a very important metal in industry. The amount of cobalt consumed in the United States in 1944 was practically six million pounds. Of this amount, stellite and stellite type alloys as well as carbide type alloys consumed 1,757,673 pounds. Apart from this, its chief use was for the production of ' Alnico ' magnets (an alloy of cobalt, nickel, and aluminium). This magnet is very powerful and capable of lifting 60 times its own weight. Cobalt is extensively used as a bonding or cementing material in the manufacture of tungsten carbide and other metal carbide tool tips and dies. These bonded carbide tools have a hardness approaching that of the diamond, and are rapidly displacing high-speed steel for cutting tools. Less important uses for cobalt are to be found in certain of its alloys, such as non-corrosive and tool steels. Minor uses for certain non-metallic purposes are to be found in ground-coat frit and pigments, and in catalysts. Cobalt oxide is a very efficient catalyst for the oxidation of ammonia, and cobalt-bearing substances are in use as catalysts for the synthetic production of gasoline. A novel and to a certain extent recent use of cobalt is in soil dressings in areas where cobalt deficiency has produced such diseases in sheep and cattle as ' bush sickness,' ' salt sickness ' or ' enzoötic marasmus.'

The increasing exposure of industrial workers to cobalt metal dust or cobalt oxide dust—which has resulted from the rapid growth of the cemented tungsten carbide industry—has drawn attention to possible injurious action, both on the skin, and after inhalation of this finely-divided material. There is a substantial amount of experimental work on the physiological action of cobalt reported in the literature, and it would appear that the toxicity of cobalt by mouth is low. Antal (1894) found that a gramme of cobalt nitrate in 1% solution had no perceptible effect on rabbits, although the same quantity in 5% solution was occasionally lethal. Chittenden and Norris (1889) also observed that the poisonous action of cobalt on rabbits is slow, and apparent only after the administration of relatively large doses. Intravenously, cobalt has been observed to cause paralysis of the extremities, enteritis and death. Le Goff (1930) found that cobalt given intravenously in man produced a marked dilatation of the blood vessels of the face, with a slight fall of blood pressure.

A well-recognized effect of the administration of cobalt salts to animals is polycythaemia, and most of the investigations relating to the physiological activity of cobalt have revolved around this phenomenon. Orten (1936) concluded that the haematopoietic activity of cobalt is the result of an increase in rate of formation of haemoglobin and erythrocytes, rather than of a passive accumulation of red cells resulting from a diminished rate of cell destruction. Frequent reference has been made to the work of Barron and Barron (1936), who state that small amounts of cobalt inhibit the respiration *in vitro* of various tissues—notably reticulocytes and bone marrow. However, in recent experiments Warren *et al.* have been unable to confirm this finding. Frost *et al.* found haematopoietic response at a minimum level of 1 mg. per kg. of body weight per day when cobalt was added to iron and copper in the diet.

It has been stated recently by Griffith and others that cysteine has a detoxifying action in cobalt poisoning. According to these investigators, poisoning from cobalt may be due to fixation and loss of sulphydryl compounds in tissues, with resulting interference with oxidative mechanisms. Cobalt has been shown to produce polycythaemia in rats (Griffith *et al.*, 1942; Orten *et al.*, 1932; de Savitsch *et al.*, 1936; Josland and McNaught 1938; Anderson *et al.*, 1940; Dorrance *et al.*, 1943), in rabbits (Barron and Barron, 1936; Kleinberg *et al.*, 1939), in ducks (Davis *et al.*, 1945), and in dogs (Frost *et al.*, 1941; Brewer, 1940; Davis, 1940; Frost *et al.*, 1941). Kent and McCance found that in man the gastro-intestinal tract is the main channel of excretion for the cobalt naturally occurring in food, and that, intravenously injected, cobalt is chiefly eliminated through the kidneys. The process of elimination of cobalt from the tissues of man is very slow. In the rat cobalt is excreted chiefly in the urine, and, according to Copp and Greenberg, most of the ingested cobalt is quickly eliminated. Using radioactive cobalt, these investigators found that the amounts distributed in tissues are very small, while the bone, and the glandular organs, such as the pancreas, liver and spleen, retain the larger amount. In an investigation made in the tungsten carbide industry, Schwartz and his associates have shown that powdered cobalt produces dermatitis. Stewart has shown that, in a patient sensitive to both cobalt and nickel, hyperaemic oedematous vesicular lesions were produced by cobalt, and that the sensitivity was somewhat less than to nickel. Haxthausen has experimentally demonstrated a hypersensitivity of the skin to cobalt, while Rabeau and Ukrainczyk have been unable to confirm the reciprocal hypersensitivity of nickel-sensitive subjects to cobalt and vice versa.

Columbium

Columbium (formerly referred to as niobium) and tantalum occur in the vanadium branch of Group V, and because of the toxic effect of vanadium, it might be anticipated that exposure at least to certain of the compounds of these metals would have somewhat similar effects. Both columbium and tantalum are steel-grey

metals having a bright metallic lustre. The similarity of chemical behaviour of the two makes quantitative separation somewhat difficult. Before the last decade neither metal had any particular importance, and both were regarded as laboratory curiosities. Columbium possesses the particular virtues of great corrosion resistance and a very high melting-point, and it is quite important at the present time as an alloy in jet-propelled airplanes, where materials are subject to terrific pressure and heat. A significant portion of the commercial output of columbium at the present time is used in the cemented carbide industry. Columbium forms a carbide which is extremely hard, and which imparts such unique properties to cemented tungsten carbide cutting tools that, in spite of the expense of columbium, it has attained commercial importance in this field as well as in that of alloy steel. There seems to have been no experimental work to indicate that exposure to columbium in any of its forms produces untoward physiological effect.

Indium

During the last few years the question of the toxicity of indium has repeatedly arisen, owing to the fact that this substance has had a number of new uses in industry. Heretofore indium had been so rare that exceedingly little had been separated and very few chemists had ever handled it. As with a number of rare metals, indium has shown such useful properties that separation of the metal and its commercial utilization have become increasingly important, particularly for plating aircraft engine bearings. These bearings of silver-lead alloy are extremely important. Silver has internal properties which resist failure due to fatigue. Externally, silver lacks the quality of oiliness needed in a good bearing surface. To fill that requirement, a thin layer of lead is applied to the silver surface. Unfortunately, lead is soluble in the organic acid present or formed in lubricating oils. Therefore indium is added—usually by electro-deposition—to the bearing surface. It prevents corrosion of the bearing surface without impairing the fatigue resistance or the bearing properties. At the same time, by increasing its wettability, indium permits the bearing surface to retain its oil film more completely. On brass-bearing surfaces protected with indium, no corrosion products were in evidence after one year of use. After deposition on the metal-bearing surface, the indium is placed in an oven or hot oil bath and given about two hours of heat treatment at a temperature of 350° F., which is slightly above the melting-point of indium. As a result of this treatment, the indium partly diffuses into the bearing metal.

In addition to the above use, indium enters into the composition of unique alloys of silver and of copper. It is a soft metal readily scratched by the thumb nail, silvery white in colour, and melting at 155° C. It is unaffected by exposure to air. It was formerly obtained from zinc ore, but is now obtained in much larger quantities from the sludge of electrolytic copper-refining vats. Within the past few years the price of this metal has dropped from thirty dollars an ounce to four dollars an ounce.

While indium is undoubtedly toxic on intravenous or subcutaneous injection in amounts of from two to six milligrammes per kilogramme of body weight, it cannot be said to have a sufficiently high toxicity rating to be of any great industrial importance (Anon. *Brit. med. J.*, 1943). No injury has followed the application of indium or its salts—such as indium chloride or indium sulphate—to the skin (McCord *et al.*, 1942–3). Ingestion experiments have shown that relatively large quantities of indium salts are without toxic effect (von Oettingen, 1932).

Osmium

With the exception of osmium, the elements of the rare metal group have not received any particular attention in industrial hygiene until lately. A compound of osmium, however, osmium tetroxide, has long been recognized as dangerous. Its nauseating odour and irritating effect on the eyes are well recognized. Deville and Debray (1859), the first to make an extended study of the elements of this group, suffered severe injury to their eyesight as a result of exposure to osmium tetroxide fume. Owing to the restricted use of osmium—and particularly of osmium tetroxide—a relatively small group of individuals has been exposed to its effects. Nonetheless, a number of cases of severe poisoning have been reported. One authentic fatal case, resulting from the inhalation of osmium tetroxide, was reported by Raymond in 1874. The effects are acute conjunctivitis, keratitis, ulcer of the cornea, bronchitis, pneumonia, and nephritis. The severity of these symptoms depends on the degree of exposure. Laboratory workers have occasionally complained of symptoms when handling osmium tetroxide. A few years ago, because of the blackening of osmium tetroxide in contact with oil and fat, an aqueous solution of osmic acid was used in England for finger-printing. However, it was soon realized that its use in contact with the skin caused dermatitis. The pathological changes produced in animals by exposure to osmium tetroxide have been recorded and discussed by Brunot; while McLaughlin, Milton, and Perry (1946) recorded the symptoms of workers refining osmiridium. In brief, the principal effects are ocular disturbances, an asthmatic condition on inhalation, and dermatitis and ulceration on skin contact.

Platinum

While the pernicious effects of osmium have been recognized, platinum has been considered relatively harmless until very recently. During the past year (1945) Hunter and his associates published the results of their extensive investigation of four platinum refineries. They found that 52 of 91 workers exposed to the dust or spray of complex salts of platinum were subject to running of the nose, sneezing, tightness of the chest, shortness of breath, cyanosis, wheezing, and cough. Thirteen of the men complained of dermatitis. None of these symptoms was apparent in the workers exposed to metallic platinum dust only, or to the complex salts of the other precious metals, including palladium. The platinum content of air samples taken at various stations throughout the plants was determined spectrographically and found to vary from 5·0 microgrammes to 70 microgrammes per cubic metre (Fothergill *et al.*, 1945). One may safely conclude, therefore, that platinum salts, whether carried as dust or mist, present an industrial hazard, and that such aerial contaminants should be carefully controlled.

Selenium

The literature on the toxic effect of ingested selenium is extensive because of the disastrous effects of small amounts of selenium in grain and herbage consumed by livestock in western states of the United States. A survey of the rural population in these same areas has disclosed a number of symptoms attributed to selenium intoxication, together with proof of urinary excretion of selenium (Smith *et al.*, 1936). McConnell has shown by means of radioactive selenium that 3 to 10% of the original amount of selenium subcutaneously injected was exhaled within 24 hours. The question has been raised whether the garlicky odour in the breath of animals which have ingested selenium compounds may not be due to a trace of tellurium as an impurity. The nature of the volatile selenium compounds exhaled has not been settled, although it is generally assumed that they are di-methyl selenides or analogous substances. Selenium sulphide is highly toxic, as shown by animal experiments (Dudley and Miller, 1936–41). In man, a concentration of 0·005 mg. per litre is intolerable, producing eye and nasal irritation. The experimental work of Dudley indicates that the pathological changes following exposure to hydrogen selenide arise from a chemical pneumonitis which may persist in a sub-acute form, together with a fatty metamorphosis of the liver which tends to decrease with time. This is accompanied by a progressive change in the spleen.

In industry, where workers have been exposed to selenium in copper refineries, a number of symptoms such as pallor, gastro-intestinal disturbances, nasal and throat irritation, garlicky odour of breath and perspiration, metallic taste, and nervousness—as well as the urinary excretion of selenium—have been reported (Dudley and Miller). While contact with metallic or amorphous selenium has not been reported as a source of skin injury, Duvoir (1937) and Pringle (1942) have reported cases of dermatitis after exposure to selenious acid and certain of its salts. The maximum allowable concentration of hydrogen selenide should be lower than the amount which produces eye or nasal irritation—i.e. less than one milligramme per cubic metre.

Tantalum

Tantalum, a rare element of Group V, is a steel-grey metal possessing a bright metallic lustre. It has a very high melting-point and is corrosion resistant. Its properties closely resemble those of columbium, so closely indeed that quantitative separation of the two is difficult. It is well known that tantalum is of use in surgery, both for sutures and for skull plates (Burke, 1940; Pudenz, 1943). Tantalum is as strong as mild steel, and is not only non-corrosive but produces no biological stimulation in contact with living tissues. The importance of tantalum in surgery is well stressed in an interesting article by Olson, who has also made the novel finding that tantalum foil, and more recently tantalum oxide dressing, is useful over burned areas—particularly with hydrofluoric acid burns, which are extremely difficult to heal. Minor uses for tantalum are its application in radar and other electronic tubes, and as a catalyst in synthetic rubber manufacture. The greater portion of the commercial output of tantalum is absorbed in the field of the cemented carbide industry. Apparently tantalum carbide imparts characteristics to carbide-cutting tools which are unique, and which make their commercial use sufficiently important to outweigh the cost of materials.

No experimental work seems to have been reported to indicate that either tantalum or its compounds produce any untoward physiological effects. Experimental work reported by Carney shows, in fact, that tantalum metal imbedded in the abdominal wall and in bones of dogs caused no physiological disturbance. The analytical separation of columbium and tantalum is particularly difficult, and the identification of either substance in small amounts is not easy, since neither forms distinctively-coloured compounds with organic reagents specific for the substance.

Tellurium

According to Nighman the production of tellurium in the United States in 1941 amounted to 224,639 pounds, while in 1944 production has dropped to 69,025 pounds, However, producer's stocks steadily increased throughout the war period. Heretofore the principal use of tellurium has been in rubber compounding, in which, it is stated, tensile strengths are maintained at elevated temperatures and particularly good resistance to heat and ageing is obtained. It has the property of hardening lead, and it improves its resistance to acids. Minute amounts added to cast iron increase the chill depth hardness and resistance to acids. Although the amount of tellurium used in making a ton of chilled iron is small, the production of chilled car wheels makes the potential requirement for tellurium in the iron industry quite large (Waitkins, Bearse, and Shutt, 1942). Tellurium is added to stainless steels to improve machineability and does not impair their corrosion resistance. Tellurium also increases the machineability of commercial bronze and is useful in alloys with tin, silver, and magnesium.

Cases of tellurium poisoning were reported as early as 1920 by Shie and Deeds. They resulted from exposure to tellurium fumes encountered as an impurity in and around blast furnaces in a silver refinery. Seven of the 13 men examined showed evidence of tellurium absorption, indicated by garlic odour of breath and urine, suppression of sweat, dryness of the mouth, and metallic taste. Three of these had a dry, itching skin, anorexia, nausea, some vomiting, and some degree of depression or somnolence. Similar findings were reported by Steinberg *et al.* in a much more recent case of industrial exposure to tellurium. These investigators recommend that the workroom concentration of tellurium fumes should be less than the range of 0·1 to 1 mg. per 10 cubic metres of air which they encountered. Apart from those noted above, severe cases of industrial injury owing to exposure to tellurium or its compounds have not been reported in the literature. This year Keall, Martin, and Tunbridge reported two fatalities resulting from the accidental injection of sodium tellurite in place of sodium iodide.

Thorium

Although the incandescent gas mantle was more important formerly than at present, a significant amount of thorium is handled industrially at the present time, not only for the manufacture of gas mantles, but as a catalytic agent, in ceramics, and in certain pharmaceutical preparations. Thorium and its salts are only slightly poisonous. However, thorium belongs to the group of radioactive substances, and several recent references point out the danger of continued contact with thorium compounds.

In the manufacture of gas mantles a mixture of thorium and cerium nitrates in the proportion of 99 to 1 is used, and from 1 to 2% of sulphuric acid is invariably added since this produces a soft, voluminous ash from six to ten times as bulky as that obtained from the pure nitrate alone. A small amount of beryllium nitrate is usually introduced, which tends to increase the hardness of the mantle. The woven material is treated with this solution, dried, and coated with collodion. In the process of preparing these mantles, workers have contracted a form of diffuse dermatitis, which was first described in this industry by Friedländer. A later case, also described by Friedländer, presented much the same features, with additional swelling of the hands and formation of deep cracks. Beyond these purely local effects, no cases of poisoning from contact with thorium compounds in industry have been reported. However, the danger of lung cancer in man following the administration of 'thorotrast' has been referred to by Bauer, and carcinogenic effects of thorium after the administration of thorium compounds have been pointed out by Roussy and Guéren. The Journal of the American Medical Association has pointed out editorially the potential hazards of the diagnostic use of thorium dioxide. Stenstrom and Vigness have shown that certain radioactive elements in the thorium series have been found in the faeces, urine and breath of two patients six and seven years after the intravenous injection of 'thorotrast.' In these cases thorium-*X* was found in the faeces, and thoron in the breath. Damage to the liver and spleen (Stewart *et al.*, 1932; Huguenin *et al.*, 1932), and fibrotic changes in regional lymph nodes (Harris and Friedrichs, 1932) have also been reported following the clinical or experimental use of thorium dioxide.

In the above discussion mesothorium has not been referred to. This substance, which is extensively used in the painting of watch and clock dials, is well known to produce severe and fatal injury (Evans, 1943). However, it is of interest that Evans and Goodman (1940), in a recent article on the thoron content of air and its relation to lung cancer, give data for the thoron content of air in a mantle manufactory; the concentration ranged from 23 to 400 times the recommended value of 10^{-11} curies per litre of air for safe working conditions.

Uranium

The enormous popular interest engendered by the release of the atomic bomb has focused attention on the possible toxicity of uranium—this attention being out of all proportion to its former industrial importance. It is of course possible that, with the very active progress

being made in the use of uranium as a source of energy, industrial exposure may be anticipated on a fairly large scale within the next few years. It should be emphasized that uranium is an extremely poisonous element. Nephritic injury similar to that produced by mercury and arsenic has long been known to result from uranium administration in animals. In general, animals poisoned with uranium show hepatic degeneration which appears to be parallel with the degree of acidosis rather than with the dose (MacNider, 1936). The chronic nephritis which occurs after the administration of uranium has been noted by a number of investigators since the first recorded study made by Leconte in 1854. Recent studies by Holman and his associates have shown that uranium produces acute necrotizing arterial lesions affecting principally the large elastic arteries. This specific injury is not confined to uranium (Holman, 1941, 1942, 1943). Although rare in man, uranium poisoning affecting four workers was reported in 1925 by de Laet. Newspaper accounts of poisoning in the manufacture of the atomic bomb have appeared, but nothing has so far been reported in the scientific literature.

Hoffmann has recently presented evidence regarding the distribution of uranium in various animal tissues; and, on the basis of this distribution, he regards it as an element essential for the life process. Holman and his associates (1944) report that sodium citrate therapy is effective in cases of uranium injury.

It should be pointed out that, since uranium hexafluoride is a volatile substance, it has practical importance in the thermal diffusion process of separation of the isotopes of uranium. Since fluorine occurs in only one atomic form, there are three possible hexafluorides of uranium. Concentration of the lighter $U\text{-}235F_6$ is accomplished by diffusion through a porous barrier. The diffusion process itself is difficult, and can only be accomplished in quantity by means of the elaborate procedure adopted during the war. Information concerning the toxicity of this substance is of importance. From its physical properties and from what is known of the toxic effects of its components, it would be anticipated that uranium hexafluoride could be very toxic.

Uranium hexafluoride fumes on exposure to the air and sublimes under reduced pressure at ordinary temperatures. It boils at 56·2° C., and has a vapour pressure of 298 mm. at 37° C. This substance, therefore, vaporizes easily, and care is required in order to avoid exposure. Chemically, uranium hexafluoride is highly reactive, vigorously attacking organic substances such as alcohol, ether, and benzene; in the last case it deposits carbon. It is highly corrosive to glass and metals, and one would anticipate a chemical pneumonitis, with possible additional toxic effects from inhalation of uranium hexafluoride fumes.

Vanadium

The increase in the use of vanadium in the United States is shown by the figures for production and consumption within recent years (*Chem. Met. Eng.*, 1945). The total amount of vanadium used in industry in 1936 was 342,720 pounds, rising in 1943 to 5,179,290 pounds (figure for domestic ore only). Since 1943 total consumption has dropped somewhat. However, it still remains high (4,113,309 pounds in 1944), and doubtless will be so for some time. Vanadium is used for making special alloy steels, and is an ingredient of the best grades of high-speed steels (Johnson and Davis, 1945). While other substances exert a degasifying and deoxidizing action which aids in the formation of solid steel, vanadium is especially valuable for this purpose. It promotes fineness of grain and causes a stable solution of carbides to form in the iron matrix. The principal advantages of vanadium, however, lie in the fatigue resistance which it imparts to the alloy, in its resistance to shock impact, and in its retention of strength or hardness when hot. Apparently there is no satisfactory substitute for vanadium in the alloy steel field. Although the bulk of vanadium is used for metallurgical purposes, it has very useful applications in industry, especially as a catalyst for various chemical processes.

In common with many of the unusual metals, vanadium has been found as a constantly occurring substance in animal and plant tissues, and Bertrand suggests that it has some physiological function. However, Daniel and Hewston, using a spectrographic method of analysis, state that if vanadium functions in this manner it must do so in concentrations of less than 5 parts per million of organic content. The statistics regarding vanadium poisoning are poor, perhaps as a result of large turnover in this industry. Exposure occurs chiefly in mining and milling the ore; and, since the bulk of our vanadium ores in the past have been imported, exposure of this type has occurred in foreign countries. Patronite is the ore chiefly imported into the U.S.A. It consists mainly of vanadium sulphide. In spite of controversial statements about vanadium poisoning, there is no question but that vanadium compounds are toxic, even such substances as the crude ore dust causing striking toxic effects. This was shown in experimental work carried out in this division a short time ago. Animals were exposed to patronite dust for short periods of time of one-half to three-quarters of an hour. These animals showed a mortality of practically 100% within two to three hours after exposure. The dust is apparently extremely irritating; it causes oedema of the lungs, so that the animals die from drowning in their own tissue fluid. The best review to date of vanadium poisoning as an occupational disease is by Symanski, who has made a careful study of nineteen cases of vanadium poisoning among metallurgical employees in Germany; while Wyers (1946) has given an account of a further ten cases. Balestra and Molfino have recently reported lung damage in labourers occupied in working over petroleum ash containing vanadium. They point out that the general picture is different from that of ordinary pneumoconiosis. The potential toxicity of vanadium, together with its increasing commercial importance, suggests that it is a substance which may eventually assume marked hygienic significance.

The above summary of our knowledge of the hygiene significance of certain metals which have begun to take an important place in industry is necessarily brief. Only such references to the literature are included as would be of special interest to the industrial hygienist, and they obviously represent only a fraction of the material available. Furthermore, it will be noted that there is an occasional disparity in the amount of reference material, owing to special interest at the present time in certain of these substances, or to the fact that extensive bibliographical material is available elsewhere. The preceding discussion is intended to give information not otherwise readily obtainable regarding a number of substances with which the industrial hygienist is likely to be concerned.

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POISONING OF FIREMEN BY IRRITANT FUMES
CADMIUM AND SULPHONATED CASTOR OIL

BY

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Introduction

Fourteen members of the Fire Brigade were affected to varying degrees as the result of the inhalation of fumes from a small burning bench in one of the machine shops of a Melbourne firm manufacturing motor parts. One of them died.

Although it was not possible to determine with certainty the relative importance of the various ingredients of the fumes or smokes in producing the effects, the cases are of considerable interest from several points of view. The amount of material consumed in the fire was small in relation to the space in the machine shop, the severity and nature of the effects produced. Cadmium was found in the lungs of the fatal case, though there was no knowledge in the early stages of the investigation of any positive source of the cadmium. The period of exposure to the fumes was short, but these fumes included heat decomposition products of sulphonated castor oil.

The first object burned was a small wooden bench which carried a lathe for machining small bearings for motor cars. The bench was approximately 7 feet long by 3 feet wide and was made from hardwood (eucalyptus) and oregon pine.

The machine shop in which this bench was situated was on the first floor and approximately 100 feet long by 60 feet wide, and 12 feet high. Only the bench, a small area of floor beneath it, and the articles on it, consisting of a small box of motor car bearings (which will be referred to later), and a tin containing about 1½ pints of cutting-fluid, were burned. The cutting-fluid had the following composition:

Mineral oil	26 per cent.
Sulphonated Castor Oil	9·6 „ „
Oleic Acid	3·6 „ „
41 per cent. Caustic Soda Solution	0·8 „ „
Water	60 „ „

The bench, which stood about half-way along the shop and about 10–12 feet from one wall, was not completely destroyed.

The majority of the firemen, all of whom were experienced men, some with more than 30 years' experience in the fire brigade, and some of whom were senior officers, considered that the effects of the fumes were different from those of fires to which they were accustomed and more severe than would have been expected from the size of the fire.

It was considered at first that the fire was a minor matter and one of the first men to enter the building approached the bench and endeavoured to put the fire out with a hand chemical extinguisher of the sodium carbonate–sulphuric acid type; but after a few minutes it was found necessary to retire from the shop and to attack the fire from outside the building by means of hoses through the first-floor windows.

The fire took place on April 27, 1945, but it was not until May 2nd that it was brought to the notice of the senior author as a result of the death of one of the firemen. It was thought at first from a description of the symptoms of the deceased and others that the cases were due to poisoning by cadmium fumes, as it was known that the firm concerned manufactured and used in their bearings soft metal alloys containing cadmium. However, it was soon found that cadmium-bearing metal had only been used in this machine shop on one occasion. This was in December 1944, when a small amount of cadmium had been melted in a furnace situated in a part of the shop partitioned off from the rest. The product of this melt was machined on two lathes at a considerable distance from the lathe bench which was burned.

The cadmium melting-plant of the firm was situated on the ground floor and in a separate building which was about 100 feet away from that in which the machine shop was situated. The melting-pots were adequately ventilated by a suction exhaust system, the duct of which discharged into the open air above the roof of the next-door building. A solid wall without windows was situated between this discharge chimney and the machine shop.

As there seemed to be no chance of cadmium fumes having been the cause of the symptoms, other sources were looked for. It was found that a can of cutting-fluid containing about one and a half pints of sulphonated castor oil had been burned in the fire. All metalwork on the machines in the shop was heavily rusted on the morning after the fire. The extent of this rusting, in the opinion of the fire brigade officials, was greater than could reasonably

be attributed to the water used to extinguish the fire. The windows of the shop, and of an office partitioned off from one corner of the shop, were heavily coated with a greyish deposit. Rubbings of this were secured and submitted to chemical and spectrographic analysis. Chemical analysis showed the presence of sulphate and traces of sulphite and cadmium. Spectrographic analysis showed the presence of iron, copper, tin, lead, and cadmium as major elements; and among minor elements antimony and nickel were found. The sulphate was probably due to sulphur trioxide caused by the heat decomposition of the sulphonated castor oil.

Spectrographic analysis of the ash from the lungs of the fatal case indicated the presence of tin, cadmium and nickel in excessive quantities. The amount of cadmium, was calculated as 4 milligrams for the two lungs.

Samples of urine were collected from cases 2–13 inclusive (about 500 c.cm. from each), concentrated, mixed, and evaporated to near dryness. The residue was tested spectrographically. A large sample of urine from case 14 (between 1 and 2 litres) was treated similarly. Unfortunately, owing to the time which had elapsed before the occurrence was brought to the author's notice, these samples were not collected until 5 days after the fire. No differences were detected between the elements present in these samples and those in the residue from normal urine treated similarly.

Although the early investigations did not reveal any source of the cadmium, it was subsequently discovered that a wooden box (about 20 × 10 × 10 inches), containing used bearings manufactured by other firms than the one in whose shop the fire occurred, was standing near the bench and was destroyed in the fire. A similar box of miscellaneous bearings derived from various makers was found; and from this, ten bearings were picked out at random and tested for cadmium. Two showed the presence of cadmium, the amount in the soft metal lining of one bearing being 96·0 per cent. There seems little doubt, therefore, that the source of the cadmium was the box of bearings which had been burned.

Case Reports

The fire had been going for a short but unknown length of time before the arrival of the fire brigade at 9.10 p.m. on Friday, April 27, 1945. All the men, with the exception of Case 1, who died, were questioned on Wednesday, May 2nd, and Thursday, May 3rd, by one of the authors (D.O.S.) who was notified of the incident on May 2nd. A number of the firemen who were concerned with the fire were subsequently present at another fire in a picture theatre on Sunday, April 29th, at 1 a.m. This second fire was fought entirely from the outside and from neighbouring buildings. Careful questioning indicated that there were no noxious fumes or smokes encountered on this occasion. Any influence attendance at this second fire could have had on the effects of the first was due to the muscular exertion involved.

Case No. 1. Fatal. Aged 56 years. This patient felt unwell at the second fire on Saturday night. On the following Sunday he was short of breath and had pain in the chest. He felt very ill on the Monday while attending court to give evidence He later had an haemoptysis and went to bed in the afternoon. He was seen by a doctor at 5 p.m. and again by another doctor between 9 and 9.30 p.m. He died at about 7 a.m. on Tuesday, May 1, 1945.

Post-mortem examination showed the following abnormal conditions:—

The heart cavities were dilated and contained fluid blood and a little red clot. The valve rings were dilated but the cusps were normal. The coronary arteries were thickened, atheromatous and narrowed. The aorta showed moderate atheroma. There were small haemorrhages in the visceral pleura of both lungs. The lungs were large, congested and showed an intense oedema in all parts. There were numerous haemorrhages in the mucous membrane of the trachea and bronchi. The mucosa throughout was congested and the lumen showed a little fluid. The liver was congested. The spleen was congested and firm. The kidneys were congested and firm. The brain and its membranes were congested. Death was due to pulmonary oedema following the inhalation of an irritant gas.

Case No. 2. Age 65 years. Arrived at the fire at 9.10 p.m. Entered the machine shop five minutes after the men who opened the shop. Symptoms were not felt at the fire. The fire was quickly subdued and this officer was back at Brigade Headquarters at 9.36 p.m. When taking off his boots he felt giddy and stayed on the floor to kick off his trousers. He then felt constriction in his chest—lack of power to take a deep breath, pains in his ribs, and icy coldness. He was shivering and his teeth were rattling. He was giddy while lying in bed. During the night he had aches in his thighs and back. In the morning he had intense thirst, but no appetite for food. Nausea, lack of appetite and cold shivers persisted until 2/5/45, together with shallow breathing.

Case No. 3. Age 53 years. Described the smoke on going into the machine shop as not thick. He immediately noticed a 'burning' sensation in his chest more intense than that caused by ordinary hot air. Intense uncontrollable coughing occurred almost at once. After reaching headquarters his breathing was restricted and painful, and he had intense thirst. He noticed marked retraction of the intercostal tissues at this time. He went to bed and had intense throbbing in the head. A 2 a.m. the pain in his chest had eased. He got up and fell to the floor with an attack of giddiness. He was sleepless, and later he again got up and retched. He felt faint on several occasions. At 7 a.m. he got up, but could not stand and went back to bed. Then he had pains in all his limbs, buttocks, and thighs, which lasted till 8 p.m. on Saturday, April 28, 1945.

Between 1 and 2 a.m. on Sunday he arrived at the other fire, and was on his feet the whole of Sunday. He had aches all over, no appetite, and a bad taste in his mouth on this day. He continued to have 'cold shivers' until the morning of 2/5/45, when all the symptoms had cleared up.

Case No. 4. Age 47 years. Entered shop at same time as case No. 2. The first symptom he noticed was a feeling of bells ringing in his ears, followed by a lack of strength in his arms. Later he had a feeling of constriction in his chest described as being 'not due to the density of the smoke.' At midnight he could not sleep—at 4 a.m. he drank about half a gallon of water. He had 'cold shivers' all night. On Saturday morning he could not rise from his bed, and had intense pain in the

spinal region for a few seconds. He had no energy all this day. He attended the other fire at 1 a.m. on the Sunday, and while there he 'retched' for 2 hours. After returning from this fire at 4 a.m. he was unable to straighten himself owing to the severe retching. He had a severe epistaxis. He was seen by a doctor at about midday on Saturday, and then complained of pain in the region of the bases of the lungs. Apparently there were no signs as he was not put off duty. On 2/5/45 his appetite was excellent but he still felt weak, and had a 'cold in the head' and dryness of his throat; the pains in his limbs had vanished.

Case No. 5. Age 40 years, had been 18 years in the fire brigade. He entered with case No. 3. He noticed symptoms as soon as he entered the machine shop. He stated that the smoke did not appear excessive in amount. It had a 'biting' effect and took his breath away. He did not feel any pressure or pain in his chest. He retired almost at once from the shop to the fresh air, when he immediately had a severe fit of coughing and felt nauseated. His breathing was then rapid and shallow.

He returned to headquarters at some time between 9.30 and 9.45 p.m., and sat in a chair until 2.30 a.m. as he had a similar feeling to when he had attacks of asthma as a small boy. He went to bed at 2.30 a.m. and slept propped up until 6.30 a.m. He had a throbbing headache throughout Saturday and Sunday. After breakfast and dinner on Saturday he vomited and he felt nauseated all day on Saturday and Sunday. This nausea lasted until the following Wednesday. He had no pains in the limbs but had pain in the epigastrium. On Wednesday he still felt light-headed if he tried to take a deep breath. He had been affected by smoke before but never to the same degree. He had not regarded the job as any worse from the point of view of smoke or fumes than some others he had attended.

Case No. 6. Age 55 years, had been 34 years in the fire brigade. This officer used a chemical extinguisher (sodium carbonate and sulphuric acid) on the fire. He described the smoke as being more pungent on the stairs leading to the first floor than at the burning bench, and stated that the smoke was very heavy. He had no symptoms while at the fire and was inclined to make light of the whole occurrence. On Saturday morning, at 6.45 a.m., he started to shave and found that he could not raise his hand to apply the shaving-brush to his face. He felt faint, sat down, and broke into a most profuse perspiration. His legs felt too weak to hold him. At about this time he started to feel soreness in the chest, his breathing being restricted. He had no pains in his limbs and no vomiting. He noticed that the veins of his arms and hands were extremely hard and felt sore. His head felt as though it were being squeezed. He had no thirst at any stage. On Tuesday he felt well in the morning, but very tired later in the day. On Wednesday he felt very miserable when he wakened. At this stage he still complained of shortness of breath on exertion, which lasted for 10 days after the occurrence.

Case No. 7. Age 30 years. This fireman was one of the first to enter the shop. He almost immediately noticed irritation of the throat and lungs—with difficulty in breathing—and a burning sensation in his chest. He vomited while at the fire. On returning to headquarters his throat and chest felt raw. The following morning and during the whole of the Saturday he had aches and pains in his back and soreness in his throat. He vomited again after breakfast. He had shivering attacks all day on Saturday. On Wednesday he stated that his chest was still sore, and he could not take a deep breath. Smoking was unpleasant.

Case No. 8. Age 31 years, had been 6 years in the fire brigade. He entered the shop with case No. 4. He started to cough immediately, and could not get his breath and had to retire in a few moments. On the Wednesday he had pain in the chest on attempting to take a deep breath and his appetite was poor. For two days after the event he had aches all over body and limbs.

Case No. 9. Age 53 years. This fireman was one of the first to enter the shop, and he noticed that there was something other than wood smoke present in the smoke. He soon retired owing to irritation of his respiratory tract by the smoke, which made him cough. He experienced no further effects other than soreness in the throat and coughing. By midday on Saturday he had recovered.

Case No. 10. Age 29 years. This fireman went in at the start of operations and experienced no immediate effects, except a few coughs, but he thought there was something unusual in the smoke. He had been at fires in paint and oil works but the smoke and fumes in this case were different from those he had previously experienced.

At 3 a.m. he had an extremely severe headache, tightness in his chest and shortness of breath. He had difficulty in taking a deep breath owing to what was described as a 'cutting short' of the breath, but no actual pain. The tightness in his chest and shortness of breath persisted throughout the Saturday. On the Sunday these effects gradually grew less, and on the Monday he felt normal except for a dry, hacking cough.

Case No. 11. Age 51 years. This fireman went in at the start of operations and immediately noticed soreness and irritation in his throat and shortness of breath. He retired for a while and then returned to the machine shop and stayed until operations ceased.

About an hour after returning to the fire station he felt unwell; he had a feeling of pressure in the head, a very sore throat and rapid breathing. After a hot bath and a drink of hot milk and brandy he went to bed and slept soundly until 1 a.m. when he awoke feeling very hot and dry and then broke into perspiration. During the day he felt drowsy and tired. On Wednesday he still suffered from shortness of breath on exertion.

Case No. 12. Age 29 years, had been 3½ years in the fire brigade. This fireman was orderly to case No. 1. He stated that the smoke caused a choking feeling and tightness in the throat and chest. He retired after about two minutes and vomited twice, which he had not previously done at a fire. He had had a severe post-orbital headache on the right side since the fire, never having had a headache previously. The headache commenced while in bed on the Friday night and had continued since. On the Saturday morning he had an ache in the throat and chest. He was short of breath on exertion, for some days after the fire.

Case No. 13. Aged 27 years, had been 6 years in the fire brigade. He entered the shop at once on arrival. He stated that the smoke smelt like burning an aluminium or a magnesium incendiary bomb. In about 5 minutes he commenced to feel nauseated and started retching, and was short-winded. He considered the smoke had quicker effect than other smokes he had encountered. After returning to the fire station he had a drink of milk and vomited some of it. He went to bed feeling short-winded and with pain on deep breathing. In the morning (Saturday) his knees were stiff and painful and he could not bend them until they had been massaged. He had a headache for 24 hours after the fire. He felt very weak but attempted to play football in the afternoon, but had to have oxygen administered owing to his breathlessness; he could not run ten yards. He wisely decided to give up the attempt to play, although a second administration of oxygen was advised, apparently with the object of enabling him to carry on the game. At 10 p.m. on Saturday night he had a severe fit of coughing and choking when going to bed. He shook all over

and was unable to drink a mouthful of water. In spite of this he turned out to the second fire, and felt very tired on his return. On Sunday he lay down all day. If he attempted to get up he nearly fell over, feeling dizzy and coughing. He felt very nauseated, but could not vomit, and had no appetite. He could not walk upstairs without holding to the rail, and was very breathless. When seen on Thursday he was much better—his appetite was then normal, but he was still breathless on exertion. He was then in bed, where he had been kept for some days by his medical adviser.

Case No. 14. Age unknown, had been 15 years in the fire brigade. This fireman was a patient in the Royal Melbourne Hospital, having been admitted as a case of an atypical pneumonia. He stated that he did not notice any particular symptoms at the fire. After returning to the fire station he felt some tightness in his chest, but had practically no cough. He was sleepless and had a severe throbbing frontal headache on the Friday night. On the Saturday he had a headache all day, and shortness of breath on slight exertion.

He went to the second fire at 1.3 a.m. on Sunday, and was short of breath when climbing the ladders. The headache was not so severe on Sunday, but on Monday it was very severe again, and there was shortness of breath even while in bed. He was seen by a medical officer on Monday night and again at 8.30 a.m. on Tuesday, and was transferred to hospital. When seen on Thursday he was improving. His cough on occasions had produced blood-stained mucus. He eventually recovered.

Consideration of Various Possible Toxic Agents

Carbon Monoxide. Two rats were found dead on the floor of the machine shop on the morning after the fire, but were unfortunately discarded. Later, two small rats were found dead in a nest behind a partition. It is probable that they died as a result of fumes from the fire. They were somewhat decomposed, but their livers were examined spectroscopically for carbon monoxide. It was absent. While it is possible that some of the firemen did suffer some effects from carbon monoxide, the nature and time of occurrence of the symptoms indicated that this gas could not have played an important part in producing them.

Nickel Carbonyl. The inhalation of this vapour causes among other effects oedema of the lungs. The possibility that nickel carbonyl might have been responsible for the effects was considered. This substance is formed by the action of carbon monoxide on freshly reduced, finely-divided nickel at ordinary temperature. It decomposes at 180° C. Hence it is not likely that nickel carbonyl, even if formed, escaped from the region of the fire into the atmosphere of the room.

Nickel was present in the wipings from the windows in small amount as compared with the cadmium. The relative amounts of nickel and cadmium present in the bearings destroyed in the fire are not known. The white metal usually machined at the bench was composed of antimony 5 per cent., copper 3·5 per cent., nickel 0·5 per cent. and tin the balance, but the compositions of the miscellaneous bearings destroyed in the box are not known.

The melting- and boiling-points of cadmium and of the metals, contained in the white metal machined in this shop, are as follows:—

	M.-P. ° C.	B.-P. ° C.
Cadmium	320·9	767
Cadmium oxide	—	1000 (Decomposes)
Copper	1083	2300
Antimony	630·5	1380
Tin	231·85	2260
Nickel	1452	2900

The presence of nickel in small amount as compared with the respective amounts of iron, copper, tin, lead, and cadmium in the window wipings is in accordance with what might be expected from the boiling-points of these metals.

Nickel was, however, detected in the lungs of the fatal case. It may have been inhaled in the form of finely-divided nickel, or its oxide. There is no available evidence (International Labour Office, 1938) of any acute effects on the lungs due to the oxide of nickel, although workers engaged in handling this compound have been exposed to this dust in considerable amount. While there is no certainty that nickel carbonyl was not the cause, or at any rate, a contributing cause of the effects experienced, it is considered unlikely that this substance could have been present in the air inhaled by the firemen.

Antimony. In the fatal case no antimony was detected in the lung. Since death took place several days after exposure, antimony may have originally been present and subsequently removed and distributed to other organs, or eliminated. There appears to be no evidence in the literature that exposure of considerable numbers of people to inhalation of antimonial dust causes acute lung damage.

Copper and Tin. In regard to these metals or their oxides there is again no evidence of acute lung damage to people who are exposed to fumes or dusts, as in bronze casting. Brass founders' ague or 'metal fume fever' is stated by Legge (1934) to be due to the zinc oxide fumes produced in the casting of brass. Casters of bronze, an alloy of copper and tin, do not suffer from this malady.

Impregnants in the Wood. The possibility that smoke and fumes may have contained toxic substances, such as fluorine compounds derived from sodium fluo-silicate used to impregnate the wood was investigated. It was found that no impregnants had been used in the wood from which the bench was constructed, so this possibility can be excluded.

Cellulosic Materials. The possibility that the effects may have been caused by the products of the

thermal decomposition or combustion of wood was considered. The conclusion that this was unlikely was based largely on the experience of the firemen. As already pointed out, these were all men experienced in fighting the varied kinds of fires which occur in a large city containing many manufacturing establishments, and some with over 30 years' service. Numbers of them had been previously affected by smoke on one or more occasions, but the opinion was very general that there was ' something different ' about the effects of the smoke on this occasion.

Easton (1945) discusses the effects produced by the vapours from burning cellulosic materials. He reports two fatal cases of pulmonary oedema due to inhalation of smoke from a burning mattress in a prison cell and a bed respectively, and suggests that in the case of the Cocoanut Grove fire, in Boston, which killed 492 and seriously injured 100, on November 28, 1942, certain non-thermal respiratory injuries were caused by irritants in the inhaled smoke which was derived chiefly from wood, cotton, and other cellulosic materials. While admitting that smoke from the burning wood may have contributed to the effects produced in the present series of cases, it is felt that other substances played a greater part.

Cadmium

A considerable number of cases of poisoning by inhalation of cadmium fumes have now been reported. Legge (1934) refers to a fatal case due to the melting of cadmium ingots in an inadequately ventilated lean-to shed, and to 2 cases due to welding in a small oil tank with electrodes containing cadmium. Prodan (1932*a*, *b*) has discussed the history of cadmium poisoning and has carried out experimental work on animals which will be referred to later. Bulmer, Rothwell, and Frankish (1938) report 1 case due to welding cadmium-plated levers in a large open shop, and 14 cases, including two deaths, due to annealing of cadmium-plated rivets in a large room. Spolyar, Keppler, and Porter (1944) report 5 cases, including one death due to the heating of cadmium-plated pipes in an open shop. Ross (1944) reports 23 cases due to ignition of cadmium dust. Wood and canvas filter frames were also burnt, so that the exposure was not only to fumes of cadmium oxide. There were no deaths in this series of cases, but several were off duty for periods from 4 weeks to 2 months. United States Public Health Service Reports (1942) discuss cadmium poisoning and list 58 cases in industry due to inhalation, with eight deaths.

From a survey of the literature it appears that the lethal dose of cadmium for man by inhalation is not known. The minimum lethal dose for man by swallowing is not known either, but Lewin (1929) reports that 30 milligrams of cadmium sulphate (equivalent to 16 milligrams of cadmium) by mouth caused increased salivation, choking attacks, persistent vomiting, abdominal pain, diarrhoea and tenesmus, and Burdach (1827) produced nausea and vomiting by 33 milligrams of the sulphate (equivalent to 18 milligrams of cadmium).

In view of these figures the finding of 4 milligrams of cadmium in the lungs of the fatal case, in which death occurred several days after exposure, must be regarded as of considerable significance. Prodan (1932*b*) exposed cats for 30 minutes to air containing cadmium fume. One cat was killed 5 hours after exposure and determinations of cadmium in its organs were made. The other cat with similar exposure, died 12 hours after exposure ceased. From the amount of cadmium found, the respiratory rate and volume, and assuming 25 per cent. retention of cadmium, it has been calculated that the concentration of cadmium in the air was 1·2 milligrams per litre. Cats exposed for 24 hours to a concentration which has been calculated to have been 0·0165 milligrams per litre did not die, but sacrificed animals showed very severe damage to the lungs.

Estimated Concentration of Cadmium Fumes. Basing our assumptions on Prodan's work with cats, it is assumed that 30 per cent. of the total cadmium retained was found in the lungs. Drinker, Thomson and Finn (1928) have shown that about 50 per cent. of inhaled zinc oxide fume is retained in the body. Using these assumptions, and assuming a reasonable breathing rate, and knowing the approximate time spent in the harmful atmosphere by the fatal case, it has been calculated that the concentration of cadmium in the air breathed was of the order of 0·14 milligrams per litre (140 milligrams per cubic metre), i.e. fourteen hundred times the permissible limit for continuous exposure. The actual amount was probably higher than this, since the calculation is based on the lung content at death, which did not take place until several days after exposure had ceased. Further, the figures for retention refer to the overall retention in the respiratory tract and do not distinguish between the amount retained in the lungs and that retained in the naso-pharynx and trachea and that swallowed.

Another estimate of the concentration of cadmium fume is based on the probable weight of cadmium volatilized (2 lb.) and the volume of the shop (72,000 cu. ft.). This gives 440 milligrams per cubic metre, assuming no air changes. In the cases reported by Bulmer 7 lb. of cadmium were volatilized into a space of 32,890 cu. ft. during a period of 90 minutes. Assuming six air changes in this time the concentration would be 562 milligrams per cubic metre. It is not suggested that there was uniform distribution of the cadmium fumes throughout the air space in either of the above cases, but the figures are given merely to afford a rough measure of comparison.

The fire was of short duration, lasting about an hour, and occurred after working hours; the doors and most of the windows were shut. The number of air changes, therefore, would have been less than would occur during working hours. If there were three air changes during the period which

elapsed from the commencement of the volatilization of the cadmium and the entry of the firemen the concentration may have been of the order of 150 milligrams per cubic metre. This compares satisfactorily with the concentration deduced from the lung content.

Discussion of Symptoms

Spolyar, Keppler, and Porter (1944), from a review of the literature in regard to 43 cases of cadmium poisoning, compiled useful tables of the frequency of symptoms and the time of their onset. In the following table their compilation is compared with

TABLE 1

Symptoms	Median Time of Occurrence of Symptom		Average Time Present investigation
	Spolyar, Keppler and Porter (1944)	Present investigation	
Irritation of throat	4–8 hours	Few minutes	Few minutes
Headache and dizziness	6–10 hours	5 hours	5¼ hours
Chills	10 hours	3 hours	5 hours
Vomiting	10 hours	6 hours	7½ hours
Cough	20 hours	5 minutes	8 hours
Pain in chest	24–36 hours	2–9½ hours	9 hours
Dyspnoea	30 hours	1½–6 hours	7 hours
Nausea		10 minutes	3½ hours
Weakness		10 hours	9¼ hours
Diarrhoea		—	—
Aches in limbs		10 hours	9 hours
Thirst		7 hours	5¾ hours
Pain in epigastrium		—	—
Smoking unpleasant		—	—
Tightness in Chest		½ hour	1½ hours
Retraction between ribs		—	½ hour
Fainting		8 hours	8 hours

The most striking differences indicated by this Table are the much more rapid onset of irritation of the throat, of cough, and of dyspnoea in the present series of cases.

TABLE 2

THE PERCENTAGE DISTRIBUTION OF SYMPTOMS

Symptoms	Spolyar, etc. (1944)		Present Investigation		Ross's (1944) Cases		
	No. reported	Per cent.	No. reported	Percentage of 13 cases unless otherwise stated	Symptoms	No. reported	Percentage of 23 cases
Irritation of throat	22	51	6	46	Constriction of throat	6	26
Pain in chest	22	51	8	57 (14 cases considered)			
Headache and dizziness	14	32	9	64 (14 cases considered)	Headache and vertigo	4	17
Cough	13	30	7	50 (14 cases considered)	Cough	9	39
Dyspnoea	11	25·6	12	85 (14 cases considered)	Dyspnoea	21	91
Vomiting	10	23·2	6	46			
Nausea	9	21	3	23	Gastric pain Nausea Vomiting	16	70
Chills	7	16·3	5	39	Ague or fume fever	14	61
Weakness	5	11·6	6	46	Prostration	16	70
Diarrhoea	3	7	—	—			
Aches in limbs	—	—	6	46			
Thirst	—	—	3	23			
Pain in Epigastrium	—	—	1	8			
Smoking unpleasant*	—	—	1	8			
Tightness in Chest	—	—	5	39	Præcordial constriction	21	91
Retraction between ribs	—	—	1	8			
Fainting	—	—	2	15	Pneumonia Irritation of eyes	1 2	4 9

* In Spolyar's (1944) own cases one complained of smoking being unpleasant, but the rest were non-smokers.

the results found in regard to the 13 firemen concerned in the present investigation. (The details of the symptoms of the fatal case are not included.)

In Bulmer's cases, which are included in the tabulation by Spolyar and his colleagues, there were, however, some individuals who suffered from irritation of the throat after only 10–30 minutes exposure, but none who suffered from this symptom after such a short exposure as produced it in our cases.

In Bulmer's (1938) cases, tightness in the chest was reported in 40 per cent., irritation of throat in 60 per cent., dyspnoea in 33 per cent., and cramps in 13 per cent. of the 15 cases. These cases were probably included in the survey by Spolyar, Keppler, and Porter (1944).

Of a group of individuals exposed to exactly the same risk, which is not rapidly fatal, not all will suffer with the same kind of symptoms nor will they be of the same severity. Where the risk differs for different individuals in the group one may expect wide differences in the percentage distribution of symptoms suffered by one group when compared with those suffered by another group exposed to different risks.

The number of subjects in the present group is small, but with this reservation it appears that there was considerably higher incidence of dyspnoea, chills and weakness than were noticed in the cases quoted in Spolyar's (1944) survey, and there was a higher incidence of dyspnoea than in Bulmer's (1938) cases. In Ross's (1944) series of cases the percentage of those who suffered from dyspnoea, gastric symptoms, and weakness was much higher than in the cases reviewed by Spolyar. This may have been due to greater concentration of cadmium fumes, or to the presence of other substances, such as the decomposition products of wood and canvas.

Evidence has already been adduced to show that the concentration of cadmium in our cases was probably not as high as occurred in Plant 2, as reported by Bulmer (1938). These differences in percentage incidence of symptoms and particularly in the time of onset of symptoms may, we think, be attributed to the presence of other irritating agents than cadmium in the air. In the cases reported by Bulmer (1938) and by Spolyar, Keppler, and Porter (1944), there appears to be no doubt that the toxic agent was cadmium oxide fume, and nothing else. In the present series of cases there were, in addition to the metallic compounds, thermal decomposition products of wood and of sulphonated castor oil.

It would appear that the fatal case was exposed to a concentration of cadmium oxide fumes intermediate between that which was fatal to cats after a half-hour's exposure, and that which caused severe lung damage to cats by an exposure for 24 hours.

Table 3 summarizes the data on cadmium exposure in this case, in some of the cases reported by Bulmer (1938), in the experimental results of Prodan (1932*b*) and Otto (1925) and the guinea-pig experiments reported later in this paper.

Cat 4 in Prodan's (1932) series, which weighed 1·7 kilos, showed 0·39 mg. of cadmium in the lung, 5 days after exposure ceased. This amount was not fatal, and would correspond to 16 mg. for a 70-kilo man, and compares with 4 mg. in the case described here. There is the possibility that the amount of cadmium found in the lungs of this man may have resulted from a shorter exposure to a higher concentration. This may be more harmful than a longer exposure to a lower concentration.

Animal Experiments

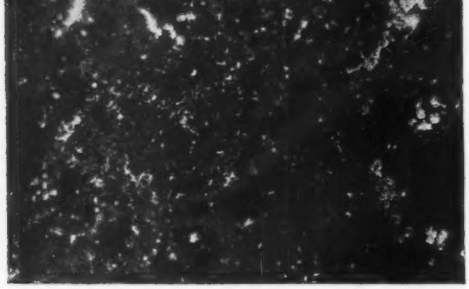
Two guinea-pigs were also exposed to cadmium oxide fumes obtained by heating in a silica tube a small amount of cadmium scraped from the motor bearings (96 per cent. cadmium). Air was passed at a known rate through a silica tube in which

TABLE 3

Subject	Concentration (Determined, or estimated approximately) (Milligrams per litre)	Time (Minutes)	Results
Cat 1 (Prodan)	1·2	30	Death
Cat 4 (Prodan)	0·018	1440	Severe lung damage
Cat (Otto)	2·0	15 (on 4 occasions)	Death
Guinea-pig	2·0	60	Death
Guinea-pig	0·4	60 on two occasions	Death
Man, Bulmer's Case 1	>0·56	90	Death
Man, Bulmer's Case 2	>0·56	90	Death
Man, Bulmer's Case 3	>0·56	30	Affected seriously
Man, Bulmer's Case 4	>0·56	10 + unknown time	Affected in less than 10 minutes. Total exposure caused serious effects
Man, Bulmer's Case 5	<0·56 (50 feet away from furnace)	20 + unknown time	Affected in less than 20 minutes. Total exposure caused serious effects
Man, Bulmer's Case 6	<0·56 (75 feet away from furnace)	5	Affected seriously
Man (present case)	0·14	20	Died.

cadmium bearing fittings were heated, gently at first, and then at bright red heat. The approximate concentration was determined from the air-flow, and the original and final weight of the cadmium.

Guinea-pig 6 was exposed for 60 minutes to a fume concentration of 2 mg. of cadmium oxide per litre. The effects were excessive salivation, and marked increase in respiration rate, but otherwise the animal appeared normal. On removal from the exposure the increased respiration rate was maintained for at least 1–2 hours, subsequently decreasing slightly. Death occurred within 24 hours. Necropsy showed the lungs to be congested and haemorrhagic, and a blood clot was in the trachea. The kidneys were enlarged, but the other organs appearing normal. Microscopically the trachea showed patches of erosion and traces of blood clot. The lungs showed large areas of emphysema, intense congestion with blood, extravasation of blood into the alveoli, fibrinous exudate in the bronchioles,

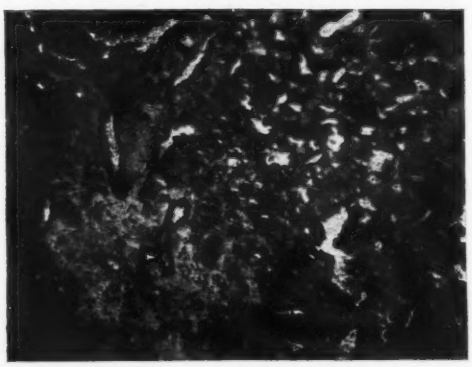


Lungs. G.-P.6. Exposed to fumes of cadmium oxide.

and a large area of red hepatization. The kidneys gave evidence of glomerulo-tubular nephritis shown by cloudy swelling in the tubules and loss of cellular outlines, congestion, and necrosis in some areas. The glomeruli showed congestion in the capillary tufts and destruction of the lining epithelium.

Guinea-pig 7 was exposed for one hour on two successive mornings to a fume concentration of 0·4 mg. of cadmium oxide per litre. During the first exposure only a slight increase in the respiration rate occurred, continuing after removal for 2 to 3 hours, and being approximately normal 24 hours later. During the second exposure the increase in the respiration rate was more marked, was maintained for a considerable time after removal from the exposure, and was faster than normal 24 hours later. Death occurred within the next 48 hours.

At necropsy the organs appeared normal. Microscopically the lungs showed widespread thickening, intense congestion and rupture of the alveolar walls, emphysema, and destruction of alveolar epithelium. In large areas the alveoli and bronchioles were blocked by fibrinous exudate. The kidneys showed cloudy swelling, and blockage of the lumen of tubules by cellular debris and fibrinous exudate, and necrosis. The glomeruli showed engorged capillary tufts, and necrotic epithelium. In some cases the lumen was completely blocked, and in others the capillary tuft was



Kidneys. G.-P.7. Exposed to fumes of cadmium oxide. The circular dots in 2, 4, 5, 6 are artefacts.

small and contracted. In some parts the glomerular structure was completely disorganized. There were patches of haemorrhage. The condition was a severe glomerulo-tubular nephritis. Table 4 summarizes the results of these animal experiments.

Effects of Decomposition Products of Sulphonated Castor Oil

At present the thermal decomposition products of sulphonated castor oil or of castor oil are not known. They include sulphur trioxide in the case of the sulphonated oil, and probably include acrolein. It has been suggested that under certain conditions alkyl sulphates, such as dimethyl sulphate, are formed. All these substances are irritant to the respiratory tract and may produce oedema of the lungs. It is well known that acrolein is a lachrymator as well as a lung irritant—the lachrymatory effect being produced by concentrations of 0·007 milligrams per litre. Ten minutes exposure to 0·35 milligrams per litre is lethal. Dimethyl sulphate is about as toxic as phosgene, and also is a powerful irritant of the conjunctivae. It is readily hydrolysed, forming sulphuric acid.

While these substances may have been present in the atmosphere, none of the firemen complained of irritation of the eyes, or of lachrymation. It might be argued that this was due to the fact that the firemen were so used to lachrymation and eye irritation caused by smoke that they hardly thought them worthy of comment. However, enquiries elicited the fact that lachrymation and eye irritation were noticeably absent or less than would have been expected from the amount of smoke.

In order to determine the effects of the products of thermal decomposition of sulphonated castor oil, guinea-pigs were exposed to the vapours produced

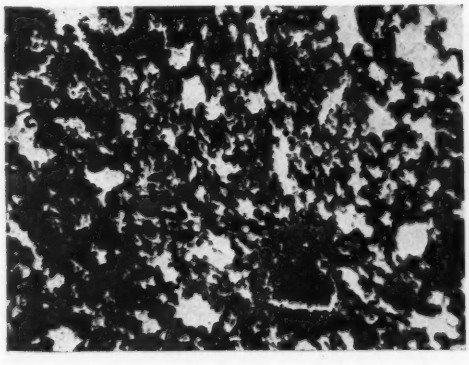
by heating small amounts of (a) the cutting-fluid, which is a dilute emulsion of sulphonated castor oil, (b) undiluted sulphonated castor oil, and (c) medicinal castor oil. Animals were exposed in a 15-litre bottle to air containing fumes let into the chamber by a side-arm from a test-tube in which a known quantity of the oil was heated. Air was drawn at a known rate, 0·7 litres per minute, over the surface of the heated liquid. In two cases the fumes were passed into the exposure-chamber through a silica tube, a few centimetres length of which was heated to about 500° C.

Guinea-pig 1. This was exposed during a period of 30 minutes to fumes caused by heating 10 c.cm. of the emulsion, the air being passed through a heated silica tube. During the period 0–15 minutes the air contained mainly water vapour and sulphuric acid mist, the fumes from the decomposed oil being evolved mainly during the 15–30 minute period. Considerable condensation of water vapour in the exposure chamber occurred. This exposure was repeated on three successive mornings. The first exposure caused sneezing, coughing and irritation of the eyes and nose and slight increase of respiratory rate. Recovery after change to fresh air was fairly rapid. The second exposure caused similar effects but they were more pronounced, and recovery was slower, the animal being very sluggish next morning.

The third exposure caused very pronounced effects; the gait was staggering, and respiration was slow, deep, audible, and sometimes of the Cheyne-Stokes type. The head was retracted and fore-feet stretched out during inspiration. The hind legs were partly paralysed. Death occurred half an hour after exposure ceased.

Necropsy showed the lungs to be emphysematous, with some patches of congestion. Microscopically, they showed intense congestion, destruction of alveolar epithelium, serous exudate, and emphysema. The epithelium of the bronchioles was necrotic. The pleura was thickened and oedematous.

Guinea-pig 2 was exposed to fumes from 2 c.cm. of heated sulphonated castor oil during thirty minutes. The effects were sneezing, coughing, which occurred almost at once, and irritation of the nose and eyes. Respiration soon became rapid and shallow but later it became slow and deep. Difficulty in standing developed, and toward the end of the experiment the animal lay prostrate. The exposure chamber was then filled with fresh air, the animal's condition improved rapidly, and respiration was normal in about half an hour. Death occurred within 24 hours.

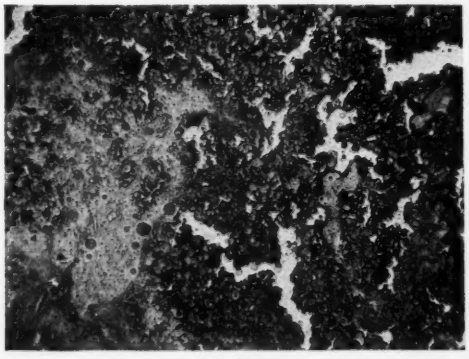


Lungs. G.-P.2. Exposed to fumes from heated sulphonated castor oil.

Necropsy showed the lungs to be congested and oedematous. Microscopically they showed intense congestion, destruction of the alveolar walls, serous exudate, and emphysema. The bronchioles showed necrosis of the epithelium. The pleura was thickened and oedematous in extensive patches.

Guinea-pig 3. This animal was exposed during one hour to air containing fumes from 1 c.cm. of heated sulphonated castor oil. The air was drawn through an asbestos-packed tower which absorbed most of the sulphur trioxide but allowed the fumes of the decomposing oil to pass. The first effects were irritation of eyes and nose, excessive salivation and very rapid respiration. Later it became slower, deeper and distressed, with some suggestion of Cheyne-Stokes breathing. There was loss of power in the hind limbs. When the jar was filled with fresh air the animal's condition improved slowly; but in the morning it was found dead.

Necropsy showed the lungs congested and there was a blood clot in the trachea. The brain showed some congestion; but other organs appeared normal. Microscopically the effects in the lungs were similar to, but more severe than, those seen in animal 2. There were patches of haemorrhage.



Lungs. G.-P.4. Exposed to fumes from heated castor oil.

The epithelium of the trachea was swollen and degenerated in places.

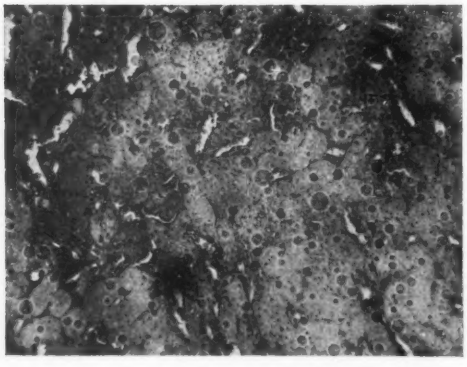
Guinea-pig 4. This animal was exposed for 60 minutes to fumes caused by the heating of 1 c.cm. of pure medicinal castor oil, the contaminated air being drawn directly into the exposure chamber. The effects were extreme irritation of the nose and eyes, increased salivation, slow, deep, and slightly

convulsive respiration, and marked physical activity. After filling the chamber with fresh air the animal became quieter and respiration gradually improved and was nearly normal after 5 hours. Next morning the animal was still alive but sluggish. It was again exposed to the same conditions. The symptoms during this second exposure were more severe—the respiration was distressed, deep and slow, and physical activity was desperate. After filling the chamber with fresh air the animal became quieter but respiration remained distressed. The animal died.

Necropsy showed that the lungs were solid. Microscopically there were large areas of consolidation, haemorrhage, oedema, emphysema, and necrosis of alveolar and bronchiolar epithelium. The pleura was thickened, haemorrhagic, and oedematous. The damage was more severe than in animals 2 and 3.

Guinea-pig 5. The conditions were the same as for guinea-pig 4 except that the heating was completed in 30 minutes. This animal showed an immediate extreme irritation of the nose and eyes and excessive salivation. Respiration was at first rapid and shallow and slightly convulsive, but later became deep, slow, and laboured. The animal showed marked activity throughout the exposure, becoming desperate towards the end. On removal from the exposure the activity ceased, but respiration continued to be very deep and distressed, and there was unsteadiness in the hind legs. Death occurred 2½ hours after the exposure ceased.

Necropsy revealed that the lungs were congested in the upper lobes, the lower being nearly normal. The brain was congested. The stomach was distended,



Kidneys. G.-P.5. Exposed to fumes from heated castor oil.

and showed haemorrhages, the wall was friable. Microscopically the lungs showed intense congestion, emphysematous patches, and necrosis of the epithelium of the alveoli and bronchioles. The kidneys showed cloudy swelling in the glomeruli, the cellular outlines being lost in many cases. The tubules showed cloudy swelling and necrosis, the central lumen being obliterated in large areas. The liver showed cloudy swelling and loss of cellular outlines in the lobules. The spleen showed cloudy swelling, and hyaline degeneration in some areas.

The main results of exposure to fumes from the cutting-emulsion, the sulphonated castor oil, and the pure castor oil were:—

(*a*) Irritation of the nose and eyes and excessive salivation, which commenced almost immediately and were severe in all cases.

(*b*) Rapid and shallow respiration with intermittent sneezing and coughing, which became deep and slow with obvious distress. Periods of apnoea and of head retraction during inspiration were also sometimes noticed in the later stages. The course of this respiratory response was similar in all cases; the severity, however, was variable. If death did not intervene temporary recovery appeared usually complete within approximately 1–2 hours.

(*c*) Effects on the activity of the animals were very different for the two different kinds of oil. The two animals exposed to the fumes from the heated sulphonated castor oil showed a strong disinclination to move, and when provoked there was a loss of power in the hind legs. In the later stages of the exposure they were lying prostrate. Recovery of activity on removal from the exposure was only partial in each case. On the other hand, the two animals exposed to the fumes from the heated medicinal castor oil showed a very marked activity, becoming desperate in the later stages of the exposure.

Guinea-pig 1 exposed to fumes from the heated emulsion which contained about 1 c.cm. of the sulphonated castor oil was less affected than the other animals, dying subsequent to the third exposure. This may have been partly due to absorption of toxic products in the water which was distilled from the emulsion, resulting in a lower concentration of toxic fumes in the air breathed.

Conclusions

From these rather brief experiments we may, perhaps, draw certain general conclusions:—

1. Exposure to these fumes in the concentration and for the times stated caused severe irritation and respiratory distress. Death resulted within a few hours. The lungs, liver and kidneys were severely damaged.
2. The effects were due mainly to the fumes resulting from the pyrolysis reactions of the vegetable oil, the sulphur trioxide fumes being only of minor significance. It was possible that alkyl sulphates played a part.
3. In addition to the respiratory damage due to such fumes, an agent was present in the fumes from the heated sulphonated oil, but not from the pure non-sulphonated oil, which was toxic to the central nervous system.

TABLE 4

Animal Number	Source of Fumes	Exposure		Results
Guinea-pig 1 ..	Cutting-emulsion 10 c.cm. containing 10 per cent. sulphonated castor oil. Fumes heated in silica tube.	Total concentration of fumes from sulphonated castor oil, approximately 50 mg. per litre. Exposure on successive days. First exposure 30 minutes. Second exposure 30 minutes. Third exposure 30 minutes.		 Affected. Affected. Death half an hour after exposure ceased.
Guinea-pig 2 ..	Sulphonated castor oil. Fumes heated in silica tube.	Sulphur trioxide; concentration increasing from 0–22 mg. per litre during 0–15 minutes, decreasing to 12 mg. per litre at the end of 30-minute period. Oil fumes: concentration increasing from 0–110 mg. per litre during 15–30-minute period.		Death within 24 hours after exposure ceased.
Guinea-pig 3 ..	Sulphonated castor oil. Fumes passed through asbestos-filled tower.	Oil fumes concentration increasing from 0–50 mg. per litre during 15–30-minute period, decreasing to 25 mg. per litre at the end of 60 minutes. Heating completed in 30 minutes.		Death within 24 hours after exposure ceased.
Guinea-pig 4 ..	Castor oil.	Oil fumes:— First Exposure: Concentration increasing from 0–25 mg. per litre during 30 minutes and from 25–33 mg. per litre at 60 minutes. Oil heated during 60 minutes. Second Exposure given 24 hours after the first. Concentration and time as for first exposure.		Affected. Death during week-end.
Guinea-pig 5 ..	Castor oil.	Oil fumes:— Oil heated during 30 minutes. Concentration increasing from 0–50 mg. per litre during 0–30-minute period, then decreasing to 25 mg. per litre at end of 60 minutes.		Death 2½ hours after exposure ceased.
		Concentration (approximate) in mg. per litre	Time in Minutes	
Guinea-pig 6 ..	Cadmium metal scrapings	2	60	Death within 24 hours after exposure ceased.
Guinea-pig 7 ..	Cadmium metal scrapings	0·4	(i) 60 (ii) 60	Slightly affected. Subsequent death during week-end.

In the case of exposure to cadmium oxide fumes the response of the animals was different from that caused by the oil fumes. This fume were much less irritant to the respiratory tract so far as the immediate effects were concerned. Weakness in the limbs and the intense activity noticed in the exposure to fumes of sulphonated castor oil and castor oil respectively, were not caused by the cadmium oxide fume. The concentrations of the cadmium oxide fume were much less than those of the oil fume, but the ultimate result was the same —death. The lungs, liver, and kidneys were all severely damaged.

It appeared probable from the animal experiments that the cadmium oxide fume was much more lethal than the fume from the cutting-compound since two exposures of one hour each to a concentration of 0·4 mg. of cadmium oxide fume per litre caused death, whereas in the case of guinea-pig 1, two exposures of thirty minutes each to a calculated concentration of 50 mg. per litre of the fumes from the sulphonated castor oil did not cause death. In the latter case the concentration of toxic fumes may have been considerably reduced by solution in the water of condensation, but it was unlikely that such effect would reduce the concentration of this toxic fume to anywhere near that of the cadmium oxide fume.

The number of animal experiments is small, but they do indicate the probabilities in regard to the fire incident. In the case of the firemen the concentration of fume from the sulphonated castor oil contained in the cutting-compound were probably of the order of 0·015 mg. per litre, or about one-tenth of the estimated concentration of the cadmium oxide fumes. It appears, therefore, that cadmium oxide fumes played the important part in producing the effects, though the early onset of irritation of the throat was more likely due to fumes from the heat decomposition of the cutting-compound than to cadmium oxide.

Summary

1. The occurrence of 14 cases of poisoning, with one death, among firemen due to inhalation of irritant fumes is described. The cadmium content of the lungs of the fatal case was 4 milligrams.

2. The probable roles of cadmium oxide fumes and those from a cutting-fluid containing sulphonated castor oil are discussed.

3. The results of experiments on the effects on guinea-pigs of the fumes of the cutting-fluid, of sulphonated castor oil, and of castor oil are described.

4. It is concluded that cadmium oxide fumes probably played an important part in producing the poisoning, the other toxic agents being derived from a cutting-compound containing sulphonated castor oil.

Acknowledgements

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THE TRANSFER OF HEAT THROUGH SUN HELMETS

BY

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1. Introduction and Summary

During the early years of the war of 1939–45, work was carried out in the laboratories of the British Cotton Industry Research Association on the heat protection afforded by military sun helmets which, at that time, were considered to be an essential article of tropical kit. Later on in the war, the use of sun helmets was discontinued, but it has been decided to publish the results of the laboratory experiments as they may be of interest in connexion with civilian helmets and also as illustrating the general method of attack on the problem of the transfer of heat through a complex system such as a sun helmet.

The basic problem was to produce a helmet which, when exposed to tropical sunshine, allowed as little as possible of the heat it received to be transmitted to the head, whilst permitting free circulation of air round the head so that moisture might evaporate and a certain amount of direct cooling of the head take place. The helmet had to be easily manufactured with little disturbance of established methods, and had to employ the minimum of scarce materials.

To achieve the minimum transmission of heat, it is important to obtain a rough idea of the relative importance of the various processes of heat transfer taking place. The most effective attack on the problem requires a knowledge of what is happening inside and outside the helmet. For instance, if we know that in an aluminium-lined helmet radiation is responsible for only a small percentage of the total heat transfer, we need not waste time in trying to reduce it further until we have examined the other modes of heat exchange. This paper describes the experimental work by which the relative importance of the various heat exchanges is determined. The flow of heat through a sun helmet is analysed into its component parts, and the way in which these combine together is worked out with the aid of an electrical analogy. Experiments are described in which the separate heat exchanges are determined, the effects on them of changes in the helmet structure are examined, and in particular, the importance of the metallic lining is brought out. The results are used to discuss the possibility of improving the helmets, and the conclusions are reached that considerable improvements could be effected by the use of a thicker, lighter material for the body of the helmet, and by increasing the size of the apertures of the top ventilator.

2. The Modes of Heat Transfer

The chief exchanges of heat are shown in fig. 1, where the letters R^1, R, C, and V stand for high temperature radiation, low temperature radiation, conduction, and convection respectively. The figure

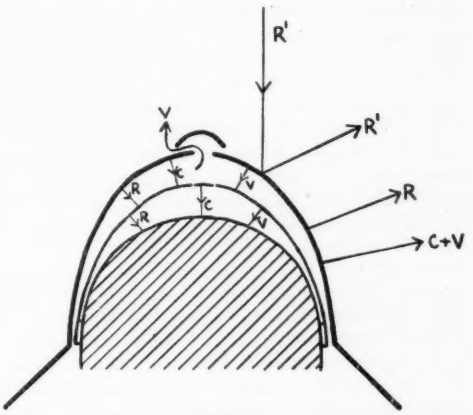

FIG. 1.—Cross-section of army sun helmet in position.

shows a helmet with dome. (The Army pattern sun helmet in existence at the time of this investigation consisted of a wool felt body stiffened with shellac, covered on the outside with khaki drill, and lined with aluminium foil or aluminized fabric. The helmet was supported away from the head by small spacers which connected it to the head band whilst allowing a gap between the latter and the helmet. Sometimes a 'drop lining' (sometimes referred to as a dome) of aluminium foil or aluminized fabric was attached to the head band as shown in the diagram.) The exchanges without a dome would be similar except that the head would take the place of the dome. No direct convection of heat from the head to the outer air is shown because the domes hitherto supplied had been practically unventilated (they had four small holes in the top, but it is doubtful whether these had much effect). No penetration of radiation through the helmet is shown, since experiments with

a sensitive thermopile failed to detect any. In the absence of a dome, or with a well-ventilated dome, there would probably be cooling of the head by evaporation of moisture followed by convection away of the vapour. Without ventilation the accumulation of vapour would soon restrict evaporation. (We are referring, of course, only to that part of the head inside the hat band.) The cooling essential to health is provided by evaporation, directly from the head through the helmet and indirectly from the body to which the overheated blood of the scalp is carried by the veins.

Let us now consider the different heat exchanges in turn.

(a) High-temperature Radiation

This is the form in which most of the heat arrives at the helmet, either as direct sunlight or as sunlight reflected from surrounding objects. Sunlight has its maximum energy (reckoned on a wave-length basis) near to a wave-length of $0{\cdot}5\mu$; most of its energy is in the visible spectrum, with roughly 5 per cent. in the ultra-violet and 20 per cent. in the infra-red, mostly fairly 'near,' i.e. with wave-lengths not greater than, say, 2μ. Only the radiation which is absorbed by the helmet goes to heat it up, and if we could cover the helmet with a perfectly reflecting material we should have no need to bother about the internal insulation. Since, however, most of the radiation concerned is in the visible range, such a helmet would appear white or metallically reflecting, and this, though permissible for civilian purposes, is ruled out on account of visibility for military use. The best that can be done is to find a fabric which is as light in colour as military considerations allow, and which reflects well in the infra-red. The latter property is desirable for a fabric which is to serve as camouflage against green vegetation, since chlorophyll reflects the near infra-red strongly. Thus there are more reasons than one for producing such a fabric. For civilian use there is no doubt that white is the best colour. A bright metallic surface would be better still, but might not be acceptable on aesthetic grounds.

(b) Low-temperature Radiation

This is the radiation exchanged between the various parts of the helmet and the head, and between the outside of the helmet and the surroundings. The energy maximum in its spectrum is in the neighbourhood of 9μ at body temperatures. There is evidence that most, if not all, textile materials are practically 'black' to these wave-lengths, i.e. they absorb and radiate to the maximum extent (Rees and Ogden, 1944 and 1946). On the other hand, many bright metal surfaces, if clean, radiate and absorb very little here, though a very thin film of organic material (e.g. grease or varnish) or of oxide is sufficient to raise the emissivity a great deal. At wave-lengths such as these the reflecting power and emissivity of a clean metal surface are determined almost solely by the electrical conductivity of the metal, the reflection being more nearly complete the higher the conductivity (Rubens and Hagen, 1903). Radiation and absorption characteristics of particular metals are not noticeable at wave-lengths longer than about 5μ. Thus silver, copper, and gold would reflect more than the aluminium which is used for lining helmets but the first two tarnish readily, whilst the third is rather expensive, even as thin foil. Tin would form a reasonable substitute for aluminium, for its emissivity in the polished state is practically the same as that of aluminium. Small differences in emissivity would have little effect on the performance of a helmet, especially when one considers that the surface is likely to get soiled in use.

The total energy radiated by a given surface is proportional to the fourth power of its absolute temperature, and the energy received by direct radiation from the surroundings (supposed to be at a uniform temperature) is proportional to the fourth power of the temperature of the solid surroundings. Thus the net rate of loss of heat by the surface considered can be written $aT_1^4 - aT_2^4$ where T_1 and T_2 are the absolute temperatures of surface and surroundings respectively. (That the coefficients of the two terms must be equal is evident from the fact that if $T_1 = T_2$ there must be no net loss of heat.) Now

$$a(T_1^4 - T_2^4) = a(T_1^2 + T^2)\,(T_1+T_2)(T_1-T_2)$$

so that, if T_1 and T_2 do not vary widely, the rate of heat transfer is approximately proportional to the temperature difference.

If we can neglect radiation reflected back and forth more than once between the surface and its surroundings, the coefficient a, and hence the rate of heat transfer, will be proportional to the product of the emissivities of the surface and its surroundings. This will be so if either the surface or surroundings are nearly 'black bodies,' and in this case the heat transfer will be proportional to the emissivity of the other surface. If both are metallic and differ appreciably from black bodies, we must take account of inter-reflections, and the heat transfer will be greater than would follow from the above approximation. We shall see that in practice it is unnecessary to have two metallic surfaces facing each other unless they are liable to get very badly contaminated. We need not, therefore, consider this case further here.

The radiated heat transfer between two surfaces inside a helmet will depend upon the extent to which one surrounds the other, but not on the distance between them, thus differing from conducted heat transfer.

(c) Convection of Heat

This includes all heat carried from one place to another by motion of the air, whether the latter is due to external causes (winds, motion of the wearer, etc.) or to circulation produced by the uneven temperature distribution. Following the usual terminology these two cases will be referred to as 'forced' and 'natural' convection respectively.

Probably a large part of the heat loss from the outer surface of the helmet is due to convection. Inside the helmet the motion of the air is more restricted and the heat transfer correspondingly smaller. The narrower the space the more restricted is the air motion, and within limits the less we should expect the convected heat transfer to be, particularly if the gap is narrow enough to prevent turbulence. Some experimental confirmation of this will be found later.

Convection has been studied quantitatively for simple shapes of surface, principally horizontal cylinders (Roberts, 1933). The results are not, of course, applicable to helmets, but we may expect that the rate of heat transfer will still be nearly proportional to the temperature difference (where this is not large) as it is in the case of the cylinders.

(d) Conduction

We shall be concerned with conduction through the material of the helmet and across the air spaces inside. With thick material like that used for pith helmets the difference in temperature between the outer and inner surfaces may be considerable, and the insulation due to the material an appreciable portion of the whole.

The transfer of heat by convection discussed in the last section involves heat exchanges between the air and solid surfaces, and, therefore, conduction of heat through the air. Thus it is not strictly permissible to consider the conduction and convection separately. None the less, it is convenient to assume as constituting the conduction a transfer of heat equal in all cases to that which would take place if the air were stationary and to ascribe any observed excess over this to convection. This arbitrary splitting up of the heat transfer is legitimate so long as we use it with discretion to obtain a qualitative picture of the process. A similar idea is used in text books on heat conduction to superpose different conduction processes, each of which could occur by itself, to give the solution to a given problem. It is there shown to be exactly legitimate for stationary media, though not, in its simple form, for moving media.

The amount of heat transferred by conduction is proportional to the temperature difference, and inversely to the thickness of the medium.

(e) Convection and Diffusion of Moisture

Considerable cooling of the bare head in hot weather may take place by evaporation of perspiration, and it is desirable that the helmet should allow this to continue. Diffusion of water vapour through air is a slow process, and, for appreciable evaporation from the head, ventilation is essential. Owing to the similarity—from the mathematical point of view—of the processes involved, it is probable that evaporation of moisture and direct cooling of the head by thermal convection will run parallel, and as the ventilation varies, the amounts of each are likely to be proportional so long as the supply of moisture at the head and other conditions remain constant.

3. The Electrical Analogy

We have now discussed the various means of heat transfer which may contribute to the total effect. Before considering how they may be determined experimentally we must consider how they combine to give the properties of the helmet as a whole. Here electrical terminology is useful. We can speak of the radiation, conduction, and convection from helmet to dome as being in parallel with each other; and of the passage from helmet to dome as being in series with that from dome to head. If we assume that the various rates of heat transfer are proportional to the temperature differences producing them, the analogy can be taken further than mere terminology. If heat flow and temperature correspond to current and potential respectively, heat flow divided by the temperature difference producing it corresponds to a conductance, and the reciprocal of this quantity to a resistance. Then we can use the usual laws of combination, adding the resistances of elements in series, and the conductances of those in parallel. We can thus draw fig. 2, which shows the 'equivalent circuit' for a helmet with dome. The letters have the same significance as in fig. 1 and are now to be taken as representing conductances. A constant current corresponding to the absorbed radiation from the sun is supposed to enter at the top of the diagram.

Batteries are shown connecting the head, the air, and the solid surroundings with 'earth' to allow for the fact that the head, air, and surroundings are at different temperatures, which are maintained, for the head, by the thermal regulation of the body, and for the air and surroundings by their large heat capacity. The 'earth' in the thermal case may be thought of as the ground at a depth where its temperature will not vary with the external conditions. When we are concerned with an artificial 'head' such as that used in our experiments, we must substitute for the head battery a conducting path representing loss of heat from the 'head.' The effects of external ventilation are shown dotted.

If the letters C_1, R_2, etc., denote the conductances of the various steps, the over-all conductance C of the helmet is given by

$$\frac{1}{C}=\frac{1}{C_1}+\frac{1}{R_2+C_2+V_2}+\frac{1}{R_3+C_3+V_3} \quad . \quad . \quad (1)$$

If the rate of supply of energy from the sun is H units, and of this a fraction α is absorbed by the helmet, we have

$$\begin{aligned}\alpha H &= C(T_O - T_H) + (C_4 + V_4)(T_O - T_A) \\ &\quad + R_4(T_O - T_S) \quad . \quad . \quad . \quad (2)\\ &= K(T_O - \theta)\end{aligned}$$

where $K = C + C_4 + V_4 + R_4$

and $$\theta = \frac{CT_H + (C_4 + V_4)T_A + R_4T_S}{C + C_4 + V_4 + R_4} \quad . \quad . \quad (3)$$

the symbols being those used on fig. 2. K may be thought of as a 'total conductance' and θ as a weighted mean of the temperatures surrounding the helmet.

Thus the temperature of the outer surface of the helmet is

$$T_O = \theta + \frac{\alpha H}{K} \quad . \quad . \quad . \quad . \quad (4)$$

and the rate of arrival of heat at the head is

$$Q = C(T_O - T) = C.\frac{\alpha H}{K} + C(\theta - T_H) \; . \quad (5)$$

The first term of this expression is the one which is of interest in connexion with protection from the sun's radiation, and it can be written

$$Q_1 = \frac{C}{C + C_E}\alpha H \quad . \quad . \quad . \quad . \quad (6)$$

where $C_E = C_4 + V_4 + R_4$, i.e. the 'External Conductance.'

To make this quantity smaller we may decrease α or C, or increase C_E, as is otherwise obvious. If, however, it should happen that C is comparable with

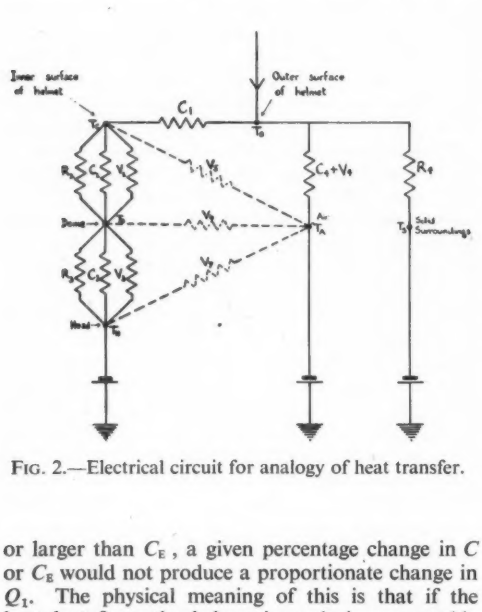

FIG. 2.—Electrical circuit for analogy of heat transfer.

or larger than C_E, a given percentage change in C or C_E would not produce a proportionate change in Q_1. The physical meaning of this is that if the heat lost from the helmet inwards is comparable with that lost outwards, increasing the internal insulation would cause the helmet temperature to rise appreciably and this would partially offset the good effect of the increase in insulation. Experimental evidence will be presented later, however, that, even with the least insulated helmet, C_E is not less than five times C. Hence, in practice, we can assume that the full benefit would be received from a decrease in C or an increase in C_E.

Examples of the numerical application of the methods described in this section will be found in later sections, particularly in the appendix, where head temperature rises are calculated for helmets lined with various materials whose emissivities had been determined with a Leslie cube and thermopile.

4. Determination of Internal Heat Exchanges

Apart from the determination of the fraction of the sunlight absorbed, which depends only upon the material with which the helmet is covered, the experiments had to be carried out with the apparatus immediately available, which was that originally set up for the empirical testing of helmets. In this the helmet rested upon a plaster of Paris 'head' and was subjected to radiation from an electric radiator placed vertically above it. The rise in temperature of the surface of the head after two hours was taken as a measure of the flow of heat to the head, and the temperatures of the various parts of the helmet could be measured by thermocouples. The separation of the various modes of heat transfer had to be effected by repeating such experiments under different conditions in which the modes operated in various proportions. The method is admittedly not entirely accurate since the head has not reached equilibrium in 2 hours, and its temperature rise in this time is not an exact measure either of the rate of passage of heat to it at that instant, or of the total amount of heat it has received in 2 hours. It would have been preferable to have kept the temperature of the head constant while measuring the heat influx. This, however, would have had to be somewhat elaborate, and since quick results were needed, use was made of the existing apparatus which had already been set up for the empirical comparison of different commercial helmets. It is considered that, in comparing helmets insulated in various ways, the temperature rise should give a rough relative measure of the rate of heat transfer. Consequently the results quoted in this report should be regarded as indicating orders of magnitude only.

With the unventilated helmet (i.e. the helmet with its 'chimney' and the space between helmet and head band stopped up) without a dome there are three internal conductances to be determined: those due to radiation, conduction, and 'natural' convection. Thus a minimum of three experiments under different conditions is necessary. These were chosen as follows:

(*a*) Helmet fabric-lined; conductance =

$$R_2 + C_2 + V_2 \quad . \quad . \quad (7)$$

(*b*) Helmet aluminium-lined; conductance =

$$\epsilon R_2 + C_2 + V_2 \quad . \quad . \quad (8)$$

(*c*) Space inside helmet filled with loose cotton lap; conductance $= C_2$ . . . . . (9)

It is assumed that the fabric lining to the helmet and the surface of the 'head' have maximum emissivity. The emissivity ϵ of the aluminium foil was determined in a separate experiment with a Leslie cube and thermopile. Since ϵ was found to be only 4 per cent., experiment (*b*) is virtually one in which only conduction and convection take part. In the third experiment it is assumed that all radiation and motion of the air are stopped and that the conduction of heat is equal to that which would take place through still air. This is in approximate accord with experiments on the thermal conductivity of

cotton laps. The total conductances (in arbitrary units) being thus observed, R_2, C_2 and V_2 can be calculated by solving the resulting simultaneous equations. For helmets with domes, this process can be applied separately to the space between crown and dome, and between dome and head. When the conductances for the unventilated helmet have been found thus, the extra effect of ventilation can be determined by repeating one or more of the experiments with the ventilating apertures open.

A series of experiments on these lines was carried out on a standard-pattern Army sun helmet made of felt, covered on the outside with a khaki drill and lined inside with 'Rexine.' When an aluminium lining was used it was stuck to the Rexine with rubber solution. Since very little of the lining materials were available and the emissivity could not be determined with the lining *in situ*, only two experiments were done in which the emissivity of the aluminium lining could be considered as known. In one of these the emissivity of the foil was found immediately before the latter was stuck into the helmet, and in the other the foil was stripped and its emissivity measured immediately after the helmet experiment. There is evidence that the emissivity of a lining may change after the helmet has been standing in the laboratory; hence in all other cases the emissivity of the lining was considered as an unknown to be determined by the helmet experiments. The 'drop lining' referred to here as the 'dome,' was, when used, fastened to the head band which was separated from the helmet by spacers leaving a gap of about $\frac{3}{8}$ in. between band and helmet. The distance between the head and dome was about 1 in. and the only ventilation for this space was given by four holes roughly $\frac{3}{16}$ in. in diameter at the top of the dome. It is doubtful if these would have much effect The helmet had a small ventilator at the top as well as the gap round the head band.

A 2-kilowatt radiator was used as a source of radiant heat, arranged in a horizontal position above the helmet so that the heat was radiated vertically downwards on to it. A thermopile was used to determine the radiation intensity at the helmet, and the required intensity to simulate tropical sunshine (about 1 cal./cm.^{2} min.) was found to be given when the distance from the top of the helmet to the radiator was 60 cm. The helmet was mounted on a plaster of Paris 'head,' to the top surface of which was stuck by means of 'Tenacitine' one junction of a thermocouple made of 34 s.w.g. wires of copper and Constantan. Similar couples were attached in the same way to the dome and helmet surfaces. The helmet was then exposed to the radiation, and the rise in temperature at the crown of the dummy head was measured at intervals for a period of 2 hours. During the test the helmet and head were shielded from extraneous draughts by a black fabric screen reaching down to the floor.

The black body temperature of the radiator was approximately 1180° K, so that its radiation did not in the least resemble sunlight. This did not matter so long as the outer cover of the helmet remained the same for all the experiments. Comparisons involving different coverings require to be done with radiation as near to sunlight in composition as possible, as described in section 7.

The results are shown in Table I. The temperatures quoted (except the air temperature) are the *increases* in temperature of the various points after 2 hours' exposure of the helmet to radiation. The helmet used was No. 101, except where otherwise stated.

Helmets A.1 and A.4 were identical with No. 101 in shape, material, and covering, but had different linings. The lining referred to as 'A1' was aluminium foil 7/1000 in. in thickness. That referred to as 'A1. Fab.' was an aluminized fabric.

Three types of domes were used in the experiments, viz.

(1) Outer surface (i.e. facing crown of helmet) fabric, inner surface. (i.e. facing head) aluminium foil.

(2) Outer surface aluminium foil, inner surface fabric.

(3) Outer and inner surfaces aluminium foil.

The three types are denoted by the symbols 'Tex-Al,' 'Al-Tex,' and 'Al-Al' respectively, in the column headed 'Dome' in Table 1.

Conductance, as stated earlier, is given by the ratio of heat flow to the temperature difference producing it. Thus, in the present case, the conductance of the helmet-head space is given by the ratio :

$$\frac{\text{Temperature rise of head}}{\text{(Temperature rise of helmet)—(Temperature rise of head)}}$$

Considering first the unventilated *helmets without dome*, we get values for the conductance of the helmet-head space as follows :—

For the Rexine-lined helmet, experiments 5, 25 and 30 give conductances:

$$\frac{24{\cdot}9}{30{\cdot}0} = 0{\cdot}83, \frac{24{\cdot}5}{24{\cdot}8} = 0{\cdot}99, \frac{27{\cdot}3}{28{\cdot}3} = 0{\cdot}96; \text{ mean} = 0{\cdot}93$$

For the aluminium foil lined helmet, experiments 14 and 42 give:

$$\frac{14{\cdot}0}{48{\cdot}6} = 0{\cdot}29 \text{ (emissivity 4 per cent.), and } \frac{16{\cdot}6}{41{\cdot}5} = 0{\cdot}40$$

(emissivity 7 per cent.); mean = 0·35.

For the helmet with the helmet-head space stuffed up, experiment 4 gives $\frac{10{\cdot}4}{62{\cdot}3} = 0{\cdot}17$

The means give the following equations in the form of (7), (8) and (9):

$$\left.\begin{aligned} R_2 + C_2 + V_2 &= 0{\cdot}93 \\ 0{\cdot}05R_2 + C_2 + V_2 &= 0{\cdot}35 \\ C_2 &= 0{\cdot}17 \end{aligned}\right\} \quad . \quad . \quad (10)$$

These give the following values for the constituent conductances of the helmet-head space:

$C_2 = 0{\cdot}17$; $V_2 = 0{\cdot}15$; $R_2 = 0{\cdot}61$; $0{\cdot}07R_2 = 0{\cdot}04$

Similarly, for the space *between helmet and dome*, where one is fitted, we have the following results for the unventilated case :—

TABLE 1

TESTS ON HELMETS IN STILL AIR

Experiment number	Lining	Dome	Temperature rises			Air Temperature °C.	Remarks
			Helmet °C.	Dome °C.	Head °C.		
Unventilated							
5	Rexine	—	54·9	—	24·9	23·0	
25	Rexine	—	49·3	—	24·5	20·5	Outer surface, 60·3°.
30	Rexine	—	55·6	—	27·3	20·0	
4	Rexine	—	72·7	—	10·4	20·5	Helmet-head space stuffed up.
8	Rexine	Tex-Al	60·6	46·9	14·4	19·8	
9	Rexine	Tex-Al	66·6	24·1	9·4	18·9	Helmet-dome space stuffed up
29	Rexine	Al-Tex	60·7	29·6	15·3	23·5	
28	Rexine	Al-Al	60·1	38·7	12·0	22·0	
14	Al	—	62·6	—	14·0	21·2	$\epsilon = 4$ per cent.
42	Al	—	58·1	—	16·6	21·0	Helmet A1 $\epsilon = 7$ per cent.
15	Al	Tex-Al	59·0	29·5	10·5	19·2	
16	Al	Al-Al	58·5	30·7	9·7	20·0	
41	Al.Fab.	—	64·0	—	16·8	19·0	
26	Al.Fab.	Tex-Al	60·6	31·9	10·6	19·8	Helmet A4
27	Al.Fab.	Al-Tex	56·9	21·5	12·5	21·7	
Ventilated							
3	Rexine	—	45·1	—	22·2	18·5	Outer surface 52°.
22	Rexine	—	50·8	—	22·7	20·0	
23	Rexine	—	50·7	—	23·0	17·7	
21	Rexine	—	43·5	—	19·6	19·5	'Chimney' opened up
24	Rexine	—	44·2	—	19·3	18·8	
7	Rexine	Tex-Al	58·9	43·4	13·4	18·5	
10	Rexine	Al-Tex	59·8	24·4	12·5	21·5	
6	Rexine	Al-Al	59·2	33·0	9·8	18·8	
11	Rexine	Al-Tex	59·1	29·0	9·0	22·0	Dome-head space stuffed up
12	Al	—	58·6	—	12·1	19·0	
13	Al	Al-Al	59·8	26·7	9·3	21·5	

With both surfaces textile, experiment 8 gives conductance = 1·05

With one surface aluminium foil and one textile, experiments 29 and 28 give conductances 0·49 and 0·56; mean value 0·53

With one surface textile and one aluminized fabric, experiments 15 and 26 give conductances 0·36 and 0·37, mean value 0·36.

With one surface aluminium foil and the other aluminized fabric, experiments 27 and 16 give values of 0·35 and 0·35; mean 0·35.

With the space between helmet and dome stuffed up, experiment 9 gives conductance = 0·22.

Now we do not know the emissivities in this case, but if we assume that in the case where two aluminium faces were concerned the radiation transfer was negligible, we can calculate the emissivities for the cases where only one aluminium surface was concerned. Thus we get

$$\left.\begin{aligned} R_2 + C_2 + V_2 &= 1{\cdot}05 \\ \epsilon R_2 + C_2 + V_2 &= {\cdot}53 \\ \epsilon_B R_2 + C_2 + V_2 &= {\cdot}36 \\ C_2 + V_2 &= {\cdot}35 \\ C_2 &= {\cdot}22 \end{aligned}\right\} \begin{aligned} &\text{(Al. lining, textile dome)} \\ &\text{(Rexine lining, Al. fabric dome)} \end{aligned} \quad \ldots (11)$$

Here ϵ_A and ϵ_B are the emissivities of the aluminium foil and aluminized fabric respectively. These equations give for the constituent conductances of the helmet-dome space the following values:

$C_2 = 0{\cdot}22$; $V_2 = 0{\cdot}13$; $R_2 = 0{\cdot}70$; $\epsilon_A R_2 = 0{\cdot}18$; $\epsilon_B R_2 = 0{\cdot}01$

from which we get values for the emissivities of the aluminium foil and aluminized fabric of 26 per cent. and 1 per cent. respectively. The former value is high, and it seems likely that the foil surface had become contaminated. Unfortunately, the foil lining was stripped and handled before this could be verified. The value of 1 per cent. for emissivity of the aluminized fabric comes from the small difference between two measured quantities, and should be taken as meaning merely that the emissivity was too small to be measured by this method.

For the space *between dome and head*, where the ventilation of the outer space would have little effect, we get the following conductances, obtained from both unventilated and ventilated experiments.

Inner surface of dome textile:—experiments 29, 27 and 10 give values for the conductance of 1·07, 1·39 and 1·05; mean 1·17.

Inner surface of dome aluminized fabric; experiments 8, 28, 15, 26, 16, 9, 7, 6 and 13 give 0·44, 0·45, 0·55, 0·50, 0·46, 0·64, 0·45, 0·42, and 0·53 respectively; mean value 0·49.

Dome-head space stuffed up: experiment 11 gives 0·45. Thus we have

$$\left.\begin{aligned} R_3 + C_3 + V_3 &= 1{\cdot}17 \\ \epsilon_B R_3 + C_3 + V_3 &= 0{\cdot}49 \\ C_3 &= 0{\cdot}45 \end{aligned}\right\} \quad . \quad . \quad . \quad . \quad (12)$$

If we take the value 6 per cent. for the emissivity of the aluminized fabric, we get the following values for the constituent conductances:

$C_3 = 0{\cdot}45$; $V_3 = 0{\cdot}02$; $R_3 = 0{\cdot}70$; $0{\cdot}06R_3 = 0{\cdot}04$.

For the conductance of the material of the helmet itself we have the reading of the temperature of the outer surface in experiment 25 as evidence, for what it is worth. The difference in temperature between the inner and outer surfaces of the felt near the top of the crown was 11° C., and this gives for the conductance of the solid part of the helmet, in the same units as before:

$$C_1 = 2{\cdot}2$$

It is difficult to be certain of the temperature of the outer surface when exposed to radiation, but this value for the conductance seems not unreasonable for a layer 0·3 cm. thick of felt, being 13 times the purely conductive part of the conductance of the air in the helmet-head space, which is roughly 15 times as thick.

All these conductances are brought together in Table 2.

TABLE 2

CONSTITUENT CONDUCTANCES OF UNVENTILATED HELMET SPACES: ARBITRARY UNITS

	Material of helmet	Helmet-head space. No dome	Helmet-dome space	Dome-head space
Conduction (C)	2·2	0·17	0·22	0·45
Convection (V)	0·0	0·15	0·13	0·02
Radiation (R) (no Al face)	0·0	0·61	0·70	0·70
Radiation (ϵR)* (one Al face)	0·0	0·04	0·04	0·04

* An emissivity of 6 per cent. is here assumed, i.e. that of the aluminized fabric when new.

It is as well here to repeat the caution that these figures should be regarded as giving the orders of magnitude only. None the less, they do not seem unreasonable when compared amongst themselves. The conduction progressively increases as we consider narrower and narrower spaces, whilst the convection decreases, as it should. The radiation remains of the same order, which is again what we should expect, since it will depend upon the degree to which the outer surface of the space considered surrounds the inner, and this does not alter much. We can apply a quantitative check to the conduction figures, for the helmet-dome space and the dome-head space in series should have the same conductive conductance as the helmet-head space, since the dome occupies a negligible volume. Adding, therefore, the reciprocals of the conduction figures for the two former spaces, and taking the reciprocal of the sum, we get 0·15, which may be compared with the experimental value of 0·17 for the helmet-head space.

5. Effect of Ventilation

So far we have only been considering those cases in which ventilation by air from outside was prevented by stopping up the 'chimney' and the gap between the head band and the helmet. All the heat exchanges involved were between helmet and head, or between helmet and dome, and dome and head. We have now to find what is the extra effect of allowing ventilation to take place.

In Table 3 are collected data from Table 1 arranged to show the effects of ventilation. Each line is concerned with one arrangement of the helmet as described in the first and second column; and the third and fourth columns show the mean rises of temperature for the corresponding unventilated and ventilated cases, respectively. The fifth column gives the differences, and it will be seen, as might be expected, that ventilation lowers the head temperature in every case. Of particular interest are the third and fourth rows, which show the results of experiments in which the little detachable cover which fits over the top ventilator had been raised so as to increase the size of the apertures. It will be seen that this has produced a marked increase in the cooling due to ventilation, a fact which suggests that a noticeable improvement in performance could be brought about by a small change in design here. The ventilator cover is detachable, so that no alteration to the helmet itself would necessarily be involved, and indeed, new covers could be fitted into existing helmets without any trouble. This is discussed later.

The ventilation may produce its effects in three ways: firstly, by direct cooling of the head or dome; secondly, by cooling the helmet and so reducing the flow of heat from it to the head; and thirdly, by giving increased opportunities for turbulence of the air inside the helmet, so increasing the flow of heat to the head. In columns 6–8 of Table 3 are shown the rises in temperature of the inner surface of the crown of the helmet in the unventilated and ventilated conditions, and the difference which shows the effect of ventilation. Column 9 is calculated from 3, 6, and 8, and shows what would be the effect on the head temperature of the decrease in helmet temperature shown in column 8 if it were acting alone, worked out on the assumption that the head temperature rise is proportional to the helmet temperature rise. Column 10, obtained by subtracting column 9 from column 5, shows that part of the effect of ventilation on head temperature which is due to direct cooling or to increase in turbulence. The figures are somewhat variable owing to their being deduced from small differences between large quantities, but one can draw the conclusion that the effects on both helmet and head are important, especially for the domeless helmet.

TABLE 3

Lining	Dome	Head rise			Helmet rise			Head cooling	
		Unventilated °C.	Ventilated °C.	Difference °C.	Unventilated °C.	Ventilated °C.	Difference °C.	Indirect °C.	Direct °C.
1	2	3	4	5	6	7	8	9	10
Rexine	—	25·6	22·6	−3·0	53·3	48·9	−4·4	−2·1	−0·9
Aluminium	—	14·0	12·1	−1·9	62·6	58·6	−4·0	−0·9	−1·0
Rexine	—	25·5	19·5	−6·1	53·3	43·8	−9·5	−4·6	−1·5
(Chimney opened)									
A1-fabric	—	16·8	10·7	−6·1	64·0	— *	—	—	—
(Chimney opened)									
Rexine	Tex-A1	14·4	13·4	−1·0	60·6	58·9	−1·7	−0·4	−0·6
Rexine	A1-Tex	15·3	12·5	−2·8	60·7	59·8	−0·9	−0·2	−2·6
Rexine	A1-A1	12·0	9·8	−2·2	60·1	59·2	−0·9	−0·2	−2·0
Aluminium	A1-A1	9·7	9·3	−0·4	58·5	59·8	+1·3	+0·2	−0·6

* An accident happened in this experiment, the helmet thermocouple coming adrift half-way through.

The conditions under which these experiments were done were rather artificial as regards ventilation, since the helmet was enclosed in a vertical tunnel of black cloth above the top of which was the source of heat. The cloth reached to the floor, however, and it was not possible to detect an appreciable updraught in the tunnel.

6. Determination of External Heat Exchanges

The external heat loss is more difficult to measure than the internal heat transfer, and in fact, it has only been possible to set a minimum to it. This, however, proved sufficient since it was found that the heat lost by the outer surface of the helmet is, as one would expect, much greater than that lost by the inner surface, and this is all that we require to know at the present. If the internal heat loss by the helmet was comparable to the external loss, an improvement in the internal insulation would result in a rise in temperature of the helmet. This would partially nullify the good effect of the increased insulation. As it is, however, the helmet temperature will not vary greatly as the internal insulation is improved.

The same helmet as used for most of the previous experiments was set up with Rexine lining and no dome, and with the ventilator and space round the head band stopped up (i.e. in the 'unventilated' state). Thermocouples were attached to the head and to the inner and outer surfaces of the top of the crown. The helmet was then exposed to radiation for 6 hours. The crown temperatures rose quickly during the first hour and at a much slower rate during the next 3 hours. During the last 2 hours there was no perceptible rise. The head temperature, on the other hand, was steadily rising the whole time, though at a diminishing rate. Thus the difference in temperature between the helmet and the head was steadily decreasing during the latter half of the run and, therefore, also the heat transfer from helmet to head. Since the heat supplied to the helmet by the radiation was constant, the diminishing loss of heat inside would produce a rise in temperature of the helmet until the increased loss outside compensated for it, and the extent of the temperature rise would depend upon the ratio of the internal and external losses. Assuming both to be proportional to the temperature differences producing them, and that T_O and T_H are the only variables, we get by differentiating (2):—

$$0 = K\frac{dT_O}{dt} - C\frac{dT_H}{dt} \quad . \quad . \quad . \quad . \quad (13)$$

or

$$\frac{K}{C} = \frac{dT_H}{dt} \Big/ \frac{dT_O}{dt}$$

or since

$$K = C + C_E$$

$$\frac{C_E}{C} = \left\{\frac{dT_H}{dt} \Big/ \frac{dT_O}{dt}\right\} - 1 \quad . \quad . \quad (14)$$

If at the time considered the helmet has not reached 'equilibrium' with the surroundings, there will be an additional rise in temperature due to this fact, so that (14) sets a minimum value for C_E/C. (In other words, we have neglected the thermal capacity of the helmet in (13) and (14).)

The results of the experiment are shown in fig. 3. It is impossible to say exactly what dT_O/dt was at the end of the experiment, but if we take the increases in temperature between 150 and 350 minutes exposure as being proportional to the temperature rates of change we find that $C_E/C \leqslant 5$. In doing this we have almost certainly underestimated the ratio. Moreover, the helmets used had a Rexine lining; an aluminium-lined helmet would show a still larger ratio. Thus we can be sure that the external heat loss is always large compared with the internal transfer of heat from helmet to head. This is all that we need to know at present, since it means that nearly the full benefit will be derived from any improvement in the internal insulation of the helmet. In the laboratory, the helmet is surrounded by air

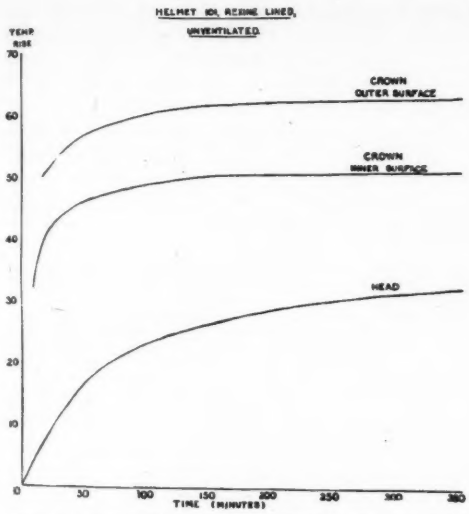

FIG. 3.—Temperature rise-time curves in ° C for helmet and plaster of Paris head.

much cooler than itself so that this large heat loss is an artificial condition. In tropical use, the surroundings will be very similar in temperature to the helmet but the relatively large conductance between them still means that the helmet temperature is little affected by changes in the internal conductance.

7. Absorption of Sunlight by Helmet Coverings

As we have seen, the ideal covering for a military helmet from a thermal point of view would be one which had the lightest permissible khaki colour, combined with the least possible absorption in the near infra-red. Experiments with the electric radiator cannot give us any idea as to the relative merits of helmet coverings, since the radiation produced is very far from being similar to sunlight. Consequently, use was made of the high-intensity carbon arc, the light from which has a spectral distribution approaching closely that of sunlight (Rees and Ogden, 1944 and 1946). Fabrics to be compared were formed into sleeves which fitted tightly over a set of similar glass tubes about 1 inch in diameter and 4 inches long. These tubes, which acted as calorimeters, were filled with water, and the bulb of a thermometer was fixed centrally in each. Several such samples wrapped round their calorimeters were placed side by side in the light from the arc, and their rates of rise of temperature were noted. Since the beam from the arc is not uniform, the samples and calorimeters were changed round so that each occupied a given position in turn and the means of the temperature rises were taken. To protect the samples from draughts they were exposed in a lidless cardboard box. The method cannot be described as accurate but it is simple and rapid, and probably good enough for the present purpose. Some results are given below for a set of fabrics as far as possible of similar thickness and structure.

Between the two extremes of aluminium foil (most efficient covering) and black cloth (least efficient) neither of which is practical for military helmets, white cloth provides the best covering from the point of view of heat insulation.

Members of H.M. Navy are thus better protected than members of the land forces, for though white coverings can be used by the Navy, they are not permitted by the land forces on account of their conspicuousness. As the results show, there is little room for improvement, however, in the coverings of the standard military type.

TABLE 4

Samples	Temperature rise, after 16 minutes' exposure
	° C.
Aluminium foil	4·8
White fabric, bleached	9·1
Light khaki, mineral dyed	11·5
Medium khaki, mineral dyed ..	11·8
Khaki fabric taken from topee ..	12·0
Medium khaki, vat dyed	13·1
Black	16·1

8. Discussion : Possibility of Improving Helmets

Before possible improvements are considered, it will be of interest to show figures for various designs of helmet that have been used in the past and have led up to the present design. In Table 5 are given the rises in temperature at the crown of the plaster of Paris head for various types of tropical helmet. The lower this rise the more efficient is the helmet. The weights of the samples are also given. In all cases the helmets are in the 'ventilated' condition, i.e. there is no interference with the system of ventilation provided by the helmet. It will be seen that the earlier helmets were lighter than the present army pattern topees, but were not so good as regards protection from the heat. This difference is mainly due to the aluminium lining used at the present time. (The first five in the above table had no metal linings.) The older helmets were also less well-ventilated than the present model, having narrower gaps between head band and crown.

In Table 6, we give similar results on British and American steel helmets both in still air and in a horizontal wind speed 2·5 miles per hour. These results are included for interest, and have no connexion with the investigation described in this report.

Now let us consider all the heat exchanges in turn with a view to deciding whether any improvement on the present helmets can be brought about. We will take them in the order in which they occur when sunlight falls on the helmet.

TABLE 5

TESTS ON HELMETS IN STILL AIR

Sample	Weight, oz.	Temperature rise after 2 hours °C.
'Lord Roberts' model (Papier maché?) ..	7·0	28·1
Ladysmith model (Papier maché?)	7·6	25·0
Cork helmet	11·3	20·3
Pith helmet	8·0	18·0
Modern felt helmet, no A1 lining	14·7	22·2
Modern felt helmet, A1 lined	14·7	11·5
Modern cork helmet, A1 lined	15·0	10·9
Modern felt helmet, A1 lined, and with A1 drop lining mounted by manufacturer	15·5	11·1
Modern felt helmet, A1 lined, but with drop lining mounted to avoid contact	15·5	9·3
Modern felt helmet, no A1 lining, but with A1 drop lining, no contact ..	15·5	9·8

TABLE 6

Helmet	Weight, oz.	Temperature rise after 2 hours	
		In still air	In wind speed of 2·5 miles per hour
		°C.	°C.
British steel helmet ..	40	27·5	5·1
British steel helmet plus Isoflex cover *	45	12·9	2·2
American steel helmet with composite lining ..	45	24·9	6·2
Lining of American steel helmet	9	30·9	7·6
Wool-felt solar topee, no A1 lining	15	22·2	3·8
Wool-felt solar topee with A1 lining	15	11·5	1·6
Wool-felt solar topee, no A1 lining but with chimney blocked up	15	26·5	5·3
Bare head	—	47·0	27·0

* 'Isoflex' is made of corrugated sheets of cellulose acetate stuck together layer upon layer, thus forming a 'honeycomb' structure. The cover referred to consists of a shell of Isoflex, roughly ¾-inch thick, shaped to fit the outside of the steel helmet. The Isoflex is enclosed between two layers of khaki drill.

(*a*) *Absorption of the Sunlight*

We have seen that a considerable diminution in the amount absorbed could be brought about by the use of a metallic or white covering, but that for military purposes, where a coloured covering is essential, there is not much room for improvement.

(*b*) *Radiation from the Outer Surface*

If this could be increased, the helmet would be cooler. For low-temperature radiation, however, most, if not all, textiles are nearly perfect radiators, so that the existing covering is already the best that can be attained in this respect.

(*c*) *Conduction and Convection from the Outer Surface*

An increase in this would be of advantage, and could be achieved by enlarging the surface area. This is done in the case of air-cooled engines by adding fins, but it is doubtful whether much could be done with a helmet without unduly complicating the construction or adding to the weight.

(*d*) *Conduction through the Helmet*

This can be, and is, cut down by increasing the thickness of the helmet, whilst keeping its conductivity low, as in the case of the pith helmet. The stiffened wool felt used in the present army sun helmets is very inefficient in its insulation for a given weight, pith being about 20 times better in this respect. The difficulty here is solely that of manufacturing under war-time conditions a helmet which is thicker without being heavier. There are a number of light 'expanded' materials available which would be suitable, but many of them are in short supply, and most would probably require more labour in the manufacture of the helmets. The shellacked felt at present employed lends itself to rapid production of the body of the helmet by a hot-pressing process. The weight of the present helmet is largely due to the desire for stiffness combined with robustness. If the former were sacrificed there would be less need for the latter, and a much lighter but equally effective helmet could be designed. It should be borne in mind, however, that stiffness in certain parts is essential in order to preserve adequate ventilation by keeping the helmet away from the head. Soft hats with the head band attached directly to the hat are poorly ventilated even if they have a few small holes punched in them. This question of ventilation is discussed later.

If it were worth while, an outer covering of Isoflex, like that designed at the Shirley Institute for steel helmets, would no doubt prove effective as an addition to the standard felt helmets, though it would add to their weight.

(*e*) *Radiation Inwards from Helmet*

This is, as we have seen, the most important means of transfer of heat in a helmet not lined with metal, but is cut down by the use of clean aluminium foil to an amount small compared with the irreducible conduction. We have not been able to test a helmet lined with aluminium which has been in use, so

that we cannot tell how far the lining retains its effectiveness under practical conditions. The use of a drop lining might help to keep the true lining clean, but this advantage is probably outweighed by the hindrance to ventilation of the head, apart from other disadvantages such as the use of valuable aluminium. An aluminium-lined topee with a double-sided aluminium drop lining is admittedly the most efficient existing system for reducing the amount of heat that reaches the head. This, however, is only part of the story. With such a topee there is almost complete lack of ventilation of the head, which, under tropical conditions, is surely a factor of the highest importance. Perspiration must be got rid of for comfort, and in a drop-lined topee there is little means of doing this, with the result that the helmet soon becomes uncomfortable to wear and may be discarded.

If we eliminate the drop lining, the helmet becomes slightly less efficient as an insulator, but is still much superior to one without the metal lining. In this connexion it should be pointed out that the high insulating efficiency of a topee fitted with a drop lining is only attained if the latter is so mounted that no contact occurs anywhere, either with the head or with the helmet itself. Those who have handled drop linings will realize how difficult it is to produce and maintain this condition. The difference in efficiency is shown in the last three lines in Table 5.

(f) Conduction from Helmet to Head

There is no practically useful substance with a thermal conductivity less than that of air. Hence the conduction of heat from helmet to head cannot be reduced below its present level.

(g) Convection of Heat from Helmet to Head

This can be stopped by filling up the space inside the helmet with a porous material, which would at the same time stop radiation without the use of metal, and whether the material was clean or not. At the same time, however, it would prevent ventilation, and there would be little, if any, gain over the present aluminium-lined helmet. Moreover, the porous material might afford convenient lodging for insects.

(h) Ventilation

This reduces the effective heat flow to the head by cooling the helmet, by direct cooling of the head, and by allowing evaporation of moisture from the head, thus also contributing to the general comfort. This is the direction in which, as it seems to us, most improvement could be brought about in the standard helmet without radical alteration in manufacture. The present helmet has taken a long step forward by introducing the wide gap between the head band and helmet body but the ventilator at the top (the 'chimney') seems to have been designed on the old principle that any hole is big enough for air.* We have already given in Table 3 some figures showing the effect of raising the ventilator cover of a standard helmet not provided with an aluminium lining. In Table 7 are shown the results of some experiments with a helmet which had such a lining. They were obtained, like the rest, on the dry plaster of Paris 'head,' and it must be remembered that, with an actual moist head, any increase in ventilation would probably show a larger effect. In the first experiment the chimney was blocked up to provide a 'zero.' (The space round the head band was left free throughout.) In the second experiment the chimney was in its normal state, and in the third the brass retaining strips were rotated about the rivet holding them so that they did not obscure the apertures. In the fourth experiment a new pattern of chimney was substituted for the usual one without any alteration to the helmet itself.

TABLE 7

State of ventilator	Temperature rise of head	Effect of ventilator
	°C.	°C.
Chimney stopped up	15·0	—
Normal state	13·9	1·1
Brass strips rotated to a new position	13·8	1·2
New pattern of chimney ..	12·4	2·6

The rotation of the brass strips was tried because it was conjectured that their position opposite to the apertures would result in an appreciable obstruction. However, this appears not to be so. It will be seen that the new pattern of chimney is more than twice as effective as the normal pattern. The former was constructed from a sheet of metal bent into the arc of a cylinder, so as to leave considerable apertures at the two sides. The present and the new pattern of chimney are shown diagrammatically in fig. 4.

To sum up, the only improvements which could be made as regards thermal insulation without radical changes in design are the substitution of thicker material and a larger ventilator cover. The former is probably impracticable as a war-time measure, and thus we are left with the ventilator as the only possibility of improvement.

If we wanted to make the best possible helmet out of the material that has passed through our hands, and irrespective of the question of quantity production, we should take the pith helmet, line it with aluminium foil, and fit it with a much larger ventilator (it has smaller apertures even than the Army pattern felt helmet). The result would be a

* The unsoundness of this principle can be seen in a general way by the following argument. With a given pressure distribution tending to produce motion of the air, the rate of flow will be inversely proportional to the resistance (i.e. dissipation of energy) brought about by surrounding matter. In the helmet this resistance will partly occur in the space between helmet and head, and partly at the chimney, and whatever the type of flow the resistance at the chimney is likely to be the greater as long as the chimney apertures are very small compared with the dimensions of the space inside the helmet.

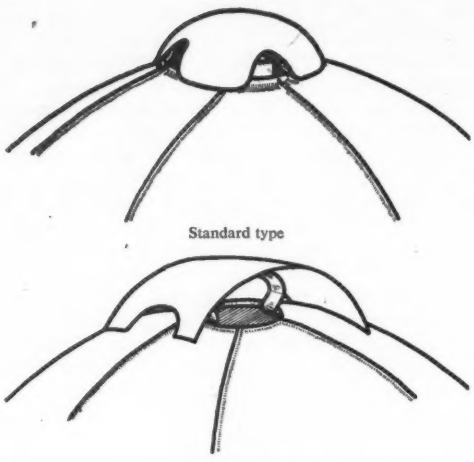

FIG. 4.—Diagrams of helmet ventilators.

great improvement on any helmet we have seen so far as good insulation with light weight goes.

9. Appendix. Emissivity of Lining Materials

In order that a lined topee shall be a good insulator, its lining must have a low emissivity, that is, it must be a poor emitter of radiant heat. The emissivity of a surface is defined as the ratio of the intensity of the radiation it emits at a given temperature to the corresponding intensity of the radiation from a perfectly black body at the same temperature. It is usually expressed as a percentage, so that if a surface has an emissivity of 100 per cent. it emits the same amount of energy as a black body under the same conditions, which is the maximum possible under those conditions.

Bright metallic surfaces have very low emissivities and, therefore, make very suitable linings for sun helmets. The ability of aluminium to retain its brightness makes it the obvious choice; indeed, the only reasonable alternative would be tin, which was probably scarcer than aluminium during the war. If foils of the same thickness were used, tin would cost about six times as much as aluminium at war-time prices.

The majority of non-metallic surfaces (including paints and textiles), no matter how white or glossy, have high emissivities, and are, therefore, unsuitable as linings. Even aluminium paint will not do, owing to the organic vehicle which covers the metal particles. Contamination of a bright metal surface by oil, grease, etc., produces a marked increase in the emissivity of the surface, as does a film of oxide in some cases (e.g., copper). The oxide film which always exists on the surface of bright aluminium does not seem to interfere appreciably.

In Table 8 are given the emissivities at 100° C. of various types of surface, measured in this laboratory by means of a Leslie cube and thermopile. In the third column are given, where possible, the temperature rises of the dummy head when covered by helmets lined with the surface in question. The fourth column is explained later.

It will be seen from these results how essential it is for the lining to be a bright, uncontaminated one. Sprayed metallic linings were found to be quite impractical, as they were very readily contaminated.

In the fourth column in Table 8 are added, as a matter of interest, temperature rises for the plaster of Paris head calculated from the emissivities given in the second column, using also the conductance data given in Table 2 and the method of combining them described in Section 3. These data leave an indeterminate constant depending upon the intensity of the radiation falling on the helmet in these experiments. This was adjusted to make the calculated value agree with the experimental (11·5° C.) in the case where the bright aluminium foil was used. Since the experimental values were found with the helmet ventilated, and the conductance calculations give values for the unventilated helmet, small corrections were applied to allow for the difference in the effect of ventilation when the head temperature rise differs from 11·5° C. The first two lines in Table 3 show a difference of 1·1° C. in the ventilation effect when the head temperature rise (unventilated) changes from 14·0° C. to 25·6° C. The corrections applied in calculating column 4 of Table 8 were assumed proportional to the difference of the head temperature rise from 11·5° C. They did not exceed 1·1° C. in any case. The agreement throughout is quite satisfactory.

TABLE 8

Sample	Emissivity at 100° C. per cent.	Temperature rise	
		Experimental ° C.	Calculated ° C.
Bright aluminium foil ..	4	11·5	(11·5)
Bright aluminium foil with a light smear of oil ..	26	—	14·7
Bright aluminium paint ..	65	19·1	19·8
Dull aluminium paint ..	74	20·6	20·8
'Sprayed' aluminium surface	25	15·7	14·7
Aluminized fabric ..	5 *	11·8 *	11·5
Aluminized fabric with 'cellophane' face ..	84	20·9	21·9
'Rexine' cloth	90–100	22·2	23–24

* The temperature rise with this helmet after it had been in store for some time rose to 13° C., presumably owing to a change in emissivity of the lining. The emissivity corresponding to this temperature rise is 13 per cent. The fabric was aluminized by the application of very thin foil, not by spraying or painting.

Acknowledgement

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AN EXPERIMENT ON THE USE OF PSYCHOLOGICAL TESTS AS AN AID TO THE REHABILITATION OF HOSPITAL PATIENTS

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Introduction

The following report is based on data which have been collected at the Birmingham Accident Hospital and Rehabilitation Centre between January and November, 1945. The work was initiated as a result of discussions between the hospital's assistant almoner and two members of the Medical Research Council's Applied Psychology Unit. These discussions centred on the applicability of vocational tests, which have been designed for subjects of normal physique, to patients attending the hospital for treatment and rehabilitation.

The Birmingham Accident Hospital treats patients who have had injuries from any cause, but the industrial accident predominates, and the emphasis in treatment is to fit the patient once more for his full work. The patient is approached as an individual and his injury related to his social background and his work. His treatment may include class work and games in the gymnasium, and work in the Occupational Therapy Department. It requires his active co-operation, and holds his interest. Most patients attend daily, and there are few absentees. Most patients, when treatment is complete, return to their old occupations, but a few—because of a residual disability, or because they were seriously ill-adjusted to their former occupation—need a change of work.

It is clear that the suitability of their choice of occupation plays an important part in the mental and physical rehabilitation of these patients. It was decided, therefore, to try out a small battery of psychological tests on patients whose future work was uncertain. Such patients, it was found, tended to fall into one of the two following classes: (*a*) those who had no ideas on the subject, (*b*) those who wanted to enter an occupation judged by the hospital authorities to be quite unsuitable.

The tests used were as follows:

1. A.H.4, a pencil and paper intelligence test in two parts. Both parts aim at testing general mental capacity and not specific aptitudes, but Part I has a verbal and arithmetical bias and Part II has a visual bias. The qualities tested are: ability to reason, to obey simple instructions, to understand the meaning of everyday words and to observe details accurately.

The subject is asked to work through examples of the test questions in his own time in order to make sure that he understands his task and to help to dissipate any nervousness induced by the test situation. Ten minutes are allowed for each part of the test proper.

2. Mechanical Ability. This is a performance test consisting of five mechanical models which are partly taken to pieces. The subject is asked to fit up as many of these models as he can in 15 minutes. He is given a screw-driver, and is told that each model is separate and complete and that the parts which are already fitted should not be dismantled. He is not informed what function the models should fulfil when they have been correctly assembled.

3. National Institute of Industrial Psychology's Form Relations Test. This is a paper and pencil test of spatial perception. The subject is required to apprehend the shapes and sizes of different figures and the relations between them.

4. Manual Dexterity

(*a*) Screw Plate.

(*b*) Rings.

In the screw plate test, the subject is given 2 minutes in which to insert as many brass screws as possible through holes drilled in the top of a screw plate. There are 29 holes in all, and inside the apparatus is a steel tube which has holes tapped in it exactly opposite the holes in the top. The subject is asked to take the holes in order, working from right to left, and to screw each screw down as far as it will go, into the inner hole. (There is a stop which prevents the subject from giving more than three or four turns, in order to avoid testing conscientiousness in addition to dexterity.)

The dexterity consists in picking up the screws, one at a time, from the standard bowl; in placing them through the top hole at a right angle to ensure that they enter the inner hole and do not fall down the side of the tube; and in giving the requisite three turns, quickly and neatly. One hand only is used for handling the screws; the other may be used to steady the screw plate.

The apparatus for the rings test consists of three key rings—one large, one medium and one small—and 35 curtain rings of slightly smaller diameter than that of the medium key ring. The subject is given the large key ring first and is asked to put on to it as many curtain rings as possible in 2 minutes. The curtain rings have to be picked up and attached one at a time, and each ring must be completely on before the next is attempted. After a short rest, the subject goes through the same procedure with the medium key ring and finally with the small one.

The score on the large key ring is ignored as its purpose is to act as a 'shock absorber' and to enable the subject to develop a technique. The sum of the scores gained on the medium and small rings is taken as the test score. Both hands are used in this test and each subject evolves his own technique, some using the curtain ring as a lever to open the key ring, some using their finger-nails, etc.

5. Alignment. The apparatus for this test consists of twenty brass rods and a box with a moveable cover. Four rows of five holes are drilled in the front plate of the box. Inside it, is another plate with a great many holes in it, including twenty which are exactly opposite the holes in the front. The distance between the two plates is slightly shorter than the length of the rods.

The subject is told that the inner plate has holes in it exactly opposite the holes in the outer plate, but he is not told of the greater number of holes in the inner plate. He is asked to pick up the rods one at a time and, beginning with the top row, to work across from left to right, placing each rod very straight through the outer hole, so that it goes into its inner hole, exactly opposite. He does this for two separate minutes, his score being the sum of the scores obtained in the 2 minutes.

Each rod correctly placed scores 3 marks; each rod slightly out in one plane scores 2 marks; each rod slightly out in both planes scores 1 mark. A rod which is placed in a hole in the inner plate, which is considerably out in one or both planes, scores 0. This test is more experimental than the other tests which were used. Its aim is to assess the subject's sense of alignment—his capacity to guide the end of a tool which he cannot see.

Not all the subjects took all these tests. In some cases of hand injury, for instance, the rings test would have been impossible. However, the tester encouraged her subjects to try as many tests as possible, believing that in some cases an objective test might detect abilities which would surprise the hospital authorities and the patient—perhaps instructing the former and encouraging the latter.

Testing procedure was, inevitably, less uniform than is permissible with groups of more normal subjects. In certain cases, for example, with the mechanical ability test, the subject had to tell the tester how to assemble the models as, owing to a hand injury, he was unable to put his own ideas into practice.

Twenty-one patients were tested in all. Testing was done by the assistant almoner, who had previously received a short course of training in giving, marking and interpreting tests at the Cambridge Psychological Laboratory. Close contact was maintained between her and the interested members of the Medical Research Council's Applied Psychology Unit throughout the investigation—which latter terminated on the tester's leaving the Birmingham Accident Hospital in November 1945.

The following report has been written by the assistant almoner who had the performance test models made for the hospital and carried out all the testing and following-up. The Appendix consists of six illustrative case histories, chosen from the collection of twenty-one.

Test Reports

The reports appended are copies of those written on the case papers of the patients concerned, and are probably less interesting in this form than in their proper context. More information was available to the writer in the form of full social case-histories and medical notes. Relevant information, particularly about follow-up, is given in the reports.

In two cases (cases 2 and 5) test results were used as a guide to further treatment and were recommended for this purpose in the second case by the surgeon, and in the first, the occupational therapist concerned. This idea seems worth developing in a rehabilitation centre where results depend very much on a patient's ability to co-operate intelligently in treatment.

In two cases (cases 2 and 6) test results shed a new light on the patient's character:

Case 2 was believed to be idle and careless. She was well-spoken and chatted in a lively way, and was believed to be intelligent. Test results suggested that this was not so, and it was thought that she would benefit by treatment requiring less mental effort. When this adjustment was made, her progress justified the change.

Case 6 was thought to be unintelligent and completely lacking in any particular talent or interest. Test results showed that he was quite intelligent, and appeared to have some ability on the mechanical side. Resettlement is now being followed on these lines, and the patient is working part-time with a firm of instrument makers while continuing treatment half the day with us.

The writer believed that case 6 was slightly 'shell-shocked' and thought that an interest in work or plans for work would help him. He has recently shown spontaneous interest and pleasure in discussing his new job, though so far in nothing else.

Case 5 insisted on taking a mechanics job against advice based on test results. He was unable to do the work, and found that he disliked it. He is now training as a gardener as he particularly wanted out-door work, and it is hoped that this will prove more satisfactory.

A patient J. N. was recommended clerical work, as he had a high leg amputation and needed work of a sedentary nature. He insisted that he disliked the idea, and was no good at clerical work, having had plenty of opportunity to find out for himself in the army. He was believed to be a capable man. He was not pressed further until test results were available, when it was found that clerical work was probably not his best line, and he was very happily resettled on instrument assembly.

This patient also found a car for himself, and altered the controls for one leg, without help or advice, so that his disability did not prevent him from driving.

In the majority of cases the tests were given to patients about whom a great deal was already known, so that information obtained in this way was largely confirmatory. In recent cases, chiefly with ex-service patients, this was not so, and it was interesting to see test results borne out in treatment.

The plan for *ex-service patients* referred for the last stage of their treatment was as follows: Treatment usually involved daily attendance in the Physiotherapy Department, the Occupational Therapy Department, and Gymnasium, and finally resettlement into suitable work. Patients were to be seen on the first attendance or as soon as possible afterwards, and problems of treatment and resettlement discussed. If resettlement was likely to be needed later, patients were to be given tests as soon as they had settled into the routine and atmosphere of the hospital. So far only cases 5, 6 and R. K., have come for testing under this scheme, and unfortunately just when the numbers of ex-service patients is increasing every week, the full scheme will have to be dropped.

General Report on giving the Tests

Every possible set-back in the form of convoys of service patients, staff shortage and ill-health has been met, and it has only been since March, 1945, that the experiment has been working satisfactorily. No difficulty has been encountered in persuading patients to do the tests, though it may take up to 2 hours of their day, and in some cases mean losing more time from work than they would normally in the course of treatment. (Patients were usually tested in time ' given ' by the Occupational Therapy Department.) Tests were explained as a normal part of the hospital treatment and the resettlement service, and they were accepted as such. The two main difficulties have been shortage of time and noise in the hospital. A whole afternoon has to be devoted to each patient, partly because more than one case rarely occurred at any time, and also because by taking patients singly it was possible to watch their reactions closely. It was found to be impossible to find a quiet room. Even when people were prevented from interrupting, and the telephone was not allowed to ring, the usual noises of the hospital seemed all-pervading. Patients did not seem to notice the noise or to be disturbed by it, though the tester was always afraid that they might be.

Specific Tests. Form Relations

This test was found most interesting, because it was apparently a test of endurance for a certain type of patient. Section 6 appeared to be the point at which some patients broke down and refused to go any further. Two patients who were temperamentally rather volatile burst into tears at this point. It was interesting to see the patients growing more and more disheartened by their inability to see the answers easily but making an effort to get answers, then, when confronted with Section 6, giving in altogether, and after this ceasing to try.

Alignment Test

The two highest scores here were obtained by cases 1 and 6, the former a good shot with a rifle, the latter a gun sighter in the Light Anti-aircraft Regiment.

This test was always given in the middle of the series, as patients seemed to enjoy doing it, and liked to have it taken to pieces afterwards. It revived interest and made a good ' break ' half-way through.

The Manual Dexterity Tests. Screw Plate

Here scores were consistently lower than they should have been on industrial grading, and they would have fitted better with a grading as follows:

Industrial	Rehabilitation Centre
AB: 20–29	AB: 15–20
C: 15–19	C: 11–14
DE: 9–14	DE: 7–10

The twenty cases tried in this grade were very well distributed, but too few to be statistically significant.

Reports obtained independently, from the Occupational Therapy Department, on every patient tested also agreed with the lower grading.

Rings Test

This test interested the writer from the point of view of the intelligence factor: did intelligence play an important part in determining test score?—had this been taken into account when using the test with normal groups?

The writer has not given the rings test often enough to assess the intelligence factor statistically, but she found that clinically patients do show signs of thinking about the test (or not thinking, according to the type of patient) in a way which is not apparent with the screw plate test. By questioning, patients, who have failed to get any rings on during the test, can be led to think of methods for themselves. It is doubtful that this knowledge would ever be enough to lift a patient out of one grade into another.

Patients with some types of finger injuries appear to be unable to handle this test at all, though they

can make a good attempt at the screw plate test. This is also true of patients who can use the fingers of one hand only.

Co-operation with Hospital Staff

Most of the assessment of a patient's ability and behaviour was done by the Occupational Therapy Department staff, and occasionally, the Gymnasium staff, who co-operated excellently and offered constructive help. There was no discrepancy found between test results and the patients' achievements in these departments, except in the two cases where there were contributory mental disorders. The medical staff also co-operated with the new service, particularly the senior surgeons, who were more often concerned with the type of patient involved. Patients were referred by the surgeon treating the patient primarily for testing, for example case 5, J. B., B. E. and C. L. Co-operation of such a satisfactory nature might be less possible where the contact between medical staff and almoners was not so well established, or where rather more conservative hopsital routine might put a brake on a new method of this kind.

Co-operation with Other Agencies

Information based on test results and recommendations suggesting suitable work were given to Ministry of Labour Resettlement Officers, and were received without question, and usually acted upon. Industrial personnel were more interested in the idea and asked questions about the tests used. The following examples of outside co-operation are recorded:

Ministry of Labour

Patient J. N. The Disablement Rehabilitation Officer of a small Labour Exchange in the country waited until he had information about the patient's abilities before deciding what type of work to find for him, and acted upon the advice given.

Industry

The Labour Manager of a firm of Scientific Instrument Makers had recently been demobilized. He was previously a colonel in the Light Anti-aircraft Regiment, and on being told the whole story of difficulties in case 6 and taking into account the test results, employed him as a part-time worker until he should be well enough to stop treatment. If he proved efficient he could continue with this work at this particular firm. The patient was by no means prepossessing, and it is doubtful whether he would have been offered the job had it not been for belief in his ability resulting from these tests, and the Labour Manager's willingness to accept them.

Other Hospitals

Case 4 had been suffering for 4 years with a hand injury severe enough to necessitate an amputation. During this time he had been unable to use the stump because of pain, and had not learned to use his left hand. On his test results it seemed worth recommending that he should be taught to write and to use his left hand, and he was sent up to the Occupational Therapy Department where he made excellent progress at first. Later it was discovered that there were psychological factors which could not be treated in this hospital, and the patient was referred to a psychiatrist. A full report was sent with him, including his test scores and report, and this information proved relevant and helpful.

Summary

Although few patients have been tested, in the writer's opinion the results have shown that this work has a place in a hospital, and that it is capable of development. The outlook of medical, auxiliary and lay staff in the hospital concerned is encouraging to experiment, and the development of work on new lines. Each patient is treated as an individual and his injury related to his social background and his work, so that the tests fitted in well with the normal plan for treatment, both in theory and practice. The organization of a service of this kind does not normally come into the duties of an Almoner's Department, and if the tests were not given by a trained psychologist, expert guidance would have to be available to supervise the work and interpret the results.

Case Reports

Case 1. D. D. aged 31.

Diagnosis. Severe fractures in several places of right femur. (Injured on Active Service.)

Left school at 14. Attended Church of England School.

Last job: Army—corporal.

Job before last: shop assistant.

General approach to test

Patient was quite co-operative though nervous. He showed no signs of distress until the end of the last test of the Form Relations group, which he was unable to do, and after this he seemed tired and not at his ease with the Mechanical Ability Test. (The Almoner wondered whether the patient was not well enough to do an hour and a half of work at this time.)

Special comments

A.H.4. Patient was better on the first part than the second, although he had no difficulty with the examples of either. He made a few mistakes but proportionately more on the second part than the first.

Manual Dexterity. He was very moderate on the Screw Plate, but good on the Rings and surprisingly good on the Alignment. Patient worked rapidly without faltering and practically every rod scored on both attempts.

Form Relations. Patient lost heart in the last test, and could not be pressed to finish the time.

Mechanical Ability. Obviously this was not the patient's strong point, and again he lost interest about three minutes before the end of the allotted time. (This might have been fatigue.) He was not pressed to finish.

Test	Score	Grade
A.H.4 (Part I)	31	B (+)
(Part II)	19	C
Total	50	C (+)
Manual Dexterity		
Screw Plate	12	DE
Ring Test	23 (20, 14+9)	AB
Alignment	41 (17+24)	A (+)
Form Relations	20	D
Mechanical Ability	11	E

Surgeon's Recommendations. He will require a sitting job.

Conclusions

Patient is quite capable and his shop-assistant's experience must have given him some opportunity to handle accounts and do clerical work. He would have to have a sitting job and he might not like full-time employment under factory conditions.

He has not a high mechanical ability score, although his manual dexterity is fairly good.

This patient might possibly do one of the lower grades of civil service job, or work as a ledger clerk or cashier. He might also be well suited to the work of telephone operator where his dexterity and his quick mental capacity would stand him in good stead.

Case 2. R. L., aged 39.
Diagnosis. Crushed right hand. Traumatic amputation of little finger.
Left school at 14.
Last job: shell band machine.
Job before last: on scrap machine.

Reason for test

Patient was referred to Almoner because she was not progressing satisfactorily in the occupational therapy department. The Occupational Therapist could not decide whether she was lazy or unintelligent. Patient had similar symptoms to P. N. but this was not borne out by her test results. She seemed more stable in her approach to the tests and less intelligent.

General approach to test

Patient is a vivacious and talkative woman, who gives the impression of being much more able than she is. Her conversation and attitude to treatment suggest a certain degree of competence which her performance belies. Her general approach to test was confident and cheerful and although she was rather depressed by the number of careless mistakes she made, she did not think her performance was unsatisfactory. Her right-hand injury seemed no disability.

Special comments

*A.H.*4. She worked through examples on her own but got nearly all of them wrong. This seemed partly carelessness and partly lack of understanding. Patient made several mistakes in the test proper, doing nearly as many questions wrong as right.

Manual Dexterity. Patient was surprisingly good considering that her hand is badly deformed and still under operative treatment. Patient enjoyed doing the tests.

Alignment. Patient was careful but not very good. Made a good many bad shots.

Test	Score	Grade
A.H.4 (Part I)	7	E
(Part II)	7	E
Total	14	E
Manual Dexterity		
Screw plate	10	DE
Ring Test	19 (14, 13+6)	D
Alignment	13 (4+9)	C

Conclusions

Patient seems capable of using her hands, provided the operation is not a difficult one, and her lively manner is no estimate of her mental ability.

Follow-up

23.10.45. In view of her long period of treatment, patient was given a holiday and then put back on simpler work, where progress was made.

Case 3. H. R., aged 31.
Diagnosis. Amputation above elbow of left arm.
Left school at 17. Attended Grammar School.
Last job: machine tool fitter and tool-room fitter.
Job before last: night entry clerk in bakery.

General approach to test

Patient was asked to do the test more from Almoner's interest and his own than for any specific resettlement problem. Patient was known to be intelligent and co-operative. He enjoyed doing the tests and had no difficulty in approaching them. Of the non-written tests only the Mechanical Ability was given, but patient was interested in the others and wanted to have them explained to him.

Special comments

*A.H.*4. He liked Part II, but did not do much better on it.

Form Relations. He did not like the last few tests but was not nervous when confronted with them. He wanted to sort out Test 8 at the end, and this was done.

Mechanical Ability. Almoner was not sure whether this should be given to patient because he might be embarrassed or discouraged by a test which should require the use of two hands. The results of A.H.4 and Form Relations suggested that it would be interesting to see patient's approach to the test as he seemed interested, and this was suggested. It was found that there was no difficulty in giving the test objectively and in timing it as for a normal patient. The tester did help to the extent of holding a screw in place and putting the links of the chain together entirely on patient's instructions, which were perfectly clear and could not be misinterpreted. Patient never hesitated, he went straight through the tests and finished them with 8½ minutes to spare.

Test	Score	Grade
A.H.4 (Part I)	46	A (+)
(Part II)	48	A (+)
Total	94	A +)
Form Relations	40	B
Mechanical Ability 8½ ..	50+17=67	A (+)

11.6.45. Patient may be found work at a plastics factory as a laboratory assistant. He is anxious to work on plastics and is going to do the City and Guild Examination as soon as he starts work.

20.6.45. Patient is to start work in the plastics laboratories on statistical control of experimental work. He is pleased with the prospect.

23.10.45. He is doing well at work. He is fitted with artificial limb.

Case 4. L. R., aged 50.
Diagnosis. Traumatic amputation all fingers right hand, reported here on 9.11.42.
Injury 2 years before—? Raynaud's disease.
Left school at 13. Attended Council School.
Last job: stamp operator.
Job before last: engine driver in engineering works.

General approach to test

Patient surprisingly stolid and settled down very well, despite his very nervous manner and his nervous approach to treatment. He grasped instructions easily and listened carefully. He did not get flustered when faced with difficulties.

Special comments

*A.H.*4. He did the examples quickly in both parts and obtained an even score on each. He had no preferences. He had some difficulty because he could only write figures with his left hand, and this only slowly and in shaky writing, but it was felt that this would not

have altered the grade of his score, which was consistent with his other results.

Form Relations. He started quite well but deteriorated at Section 6. He did not lose interest.

Manual Dexterity., As patient does not use his right hand and cannot write with his left hand it was impossible to give the Ring Test.

Screw Plate. He tackled this comfortably, without faltering, and although his score was not high, he seemed potentially capable of using his hand. He obtained a good result on his Alignment. (He develops a functional tremor of his left hand when using it consciously.)

Test	Scores	Grades
A.H.4 (Part I)	20	C
(Part II)	24	C
Total	44	C
Form Relations	22	D
Manual Dexterity		
Screw Plate	9	DE
Alignment	21 (9+12)	B
Mechanical Ability	—	—

Special Recommendations. Refer to Occupational Therapy Department in order to teach this patient to use his left hand to write.

11.6.45. Patient made great strides in the Occupational Therapy Department and quickly learnt to write with his left hand. He wrote letters to his sons in the Army who had not heard from him personally for three or four years. As soon as the question of work was approached patient began to go downhill. He seemed disinclined to face the problem.

Finally, it was suggested that patient should obtain a job as cloak-room attendant and general attendant to the Gymnasium and Massage Departments. This job would not be heavy, but would entail responsibility, the care of money and people's personal belongings. Patient seemed inclined to consider this favourably.

30.7.45. The patient was no longer co-operating satisfactorily in treatment and his condition was diagnosed by a psychiatrist as an involution depression superimposed on a traumatic neurosis.

Case 5. F. T., aged 21.

Diagnosis. Ulnar nerve palsy of left hand (gun-shot wound).

Left school at 14. Attended Senior School.

Last job: private in infantry. (No trade.)

Job before last: labourer and guillotine saw operator.

General approach to test

Patient is a keen but not a reliable worker. He makes many careless mistakes, and does not think what he is about to do. His injury seems to be no disability as far as the Manual Dexterity Tests are concerned. The patient is illiterate. He can only just spell his own name and cannot read or make sense of a difficult sentence.

Special comments

*A.H.*4. He was very slow in Part I, but quicker in Part II. Patient was better at this part, although he took as long with the examples. Several of the longer words in the first test had to be substituted by simpler ones as patient was unable to read them or understand them.

Manual Dexterity. Patient was too impetuous and not careful. This showed particularly in the Alignment Test where he got nearly all the rods in and practically none of them right.

Mechanical Ability. Patient had no idea what he was doing with any of the models except the lock, on which he got the tongue in place and seemed to know vaguely what he was doing.

Test	Score	Grade
A.H.4 (Part I)	4	E
(Part II)	13	D
Total	17	D (−)
Manual Dexterity		
Screw Plate	14	DE
Ring Test	13 (9, 8+5)	C
Alignment	2 (1+1)	E
Mechanical Ability	4	E

Comments. Patient's one ambition in life is to be a mechanic.

11.6.45. Patient found work as an unskilled mechanic in a transport garage. He says he likes the work very much. It is only a small firm but they take an interest in him and allow him to come for treatment. He has been seen at the Peripheral Nerve Injury Centre and his full disability pension is being continued.

30.7.45. Garage work was found to be unsatisfactory. Patient decided he did not like it. After some discussion he was recommended for training as a gardener, and is to go to a residential training centre for the course.

23.10.45. Now at training centre doing a gardening course.

Case 6. P. L., aged 28. 17.9.1945.

Diagnosis. Gun-shot wound left upper arm and left buttock. Has had a fracture of the left humerus. Muscular spiral palsy.

Last job: gunner, R.A.

Job before last: builder's labourer.

General Remarks. The patient was singularly disinterested in his future, but said he would like to do a course of carpentry and joinery. The almoner wondered whether he was suffering from 'shell shock.' He did not give the impression of ability or intelligence, but test results did not confirm this.

General approach to test

He tackled the tests methodically; and seemed to enjoy some of them. He had done army tests before.

Special Comments

*A.H.*4. He was slow on the first part, but surprisingly good on the second part and enjoyed it.

Manual Dexterity. He had the idea (which had been met before) that to open the key ring with the finger-nail would spoil the spring. (This was discovered after the test had been given.)

In view of the encouraging results on A.H.4 and the proposed joinery and carpentry training course, the Form Relations Test was given later.

Test	Score	Grade
A.H.4 (Part I)	13	D
(Part II)	29	B
Total	42	C
Manual Dexterity		
Screw Plate	16	C
Ring Test	3 (3+0)	DE
Alignment	34 (21+11)	A
Mechanical Ability	14 (2+10+2)	D

Follow-up

23.10.45. The patient was found temporary part-time work on instrument assembly until his arm was fit for the carpentry course; but character difficulties seemed likely to upset resettlement. As this man was one of the last patients tested, no further information was available at the time of writing the report.

ACUTE PARANITRANILINE POISONING

BY

A. ANDERSON

From The Anglo-Iranian Oil Company, Ltd., Iran.

Paranitraniline (para-nitro-amido-benzene) has not been the subject of extensive toxicological investigation although it has been included in several inquiries into the properties of the aniline group. It occurs in the form of a fine yellow powder and is employed in the manufacture of dyes and explosives and in the textile industry. Kobert and Rambousek (1913) state that the para-isomer is more toxic than the meta-, though Lewis *et al.* (1920) found the meta- to be the more dangerous; while Hunter (1944) considers that paranitraniline is more toxic than aniline itself. The modes of entry into the body are by inhalation and by absorption through the skin, and either appears to be enhanced by a moist, warm environment. Brandt (1943) puts the maximum allowable concentration in air for aniline during an 8-hour exposure at 5 p.p.m., but it is known that nitration of an aromatic compound increases its toxicity, and, therefore, the allowable concentration for paranitraniline should be less than this figure.

Symptomatology and Treatment

The symptoms of acute paranitraniline poisoning begin with headache, flushing of the face, restriction of breathing, and occasionally nausea and vomiting. If exposure continues the headache becomes violent, and tinnitus, vertigo, and extreme respiratory distress may ensue. There is generalized muscular weakness, somnolence, irritability, and an intense violet-coloured cyanosis, especially of the ears, lips, nose, tongue, fingers, and toes. After the lapse of some hours a yellowish pigmentation of the soles of the feet and the palms of the hands appears, spreading gradually to other parts of the body. If coma supervenes the case is in grave danger, and death may occur in a convulsive state. The pulse is rapid and thready, the respiration rate raised, while the temperature and blood pressure tend to be lowered in the early stages. Methaemoglobinaemia is the essential pathological change, and Hamblin and Mangelsdorff (1938), over a period of two years, found a constant correlation between the concentration of methaemoglobin in the blood and the severity of the clinical syndrome.

The cardinal principles in the treatment of acute paranitraniline poisoning are removal from contact, removal of the contaminated clothing, and bathing the patient. It is of the utmost importance that all clothing should be removed as soon as possible and that bathing is thorough, otherwise absorption via the skin will continue. Oxygen is of considerable assistance, but it will be found that the mild or moderately severe case tends to recover spontaneously. Rest is essential and exertion should be avoided as this markedly increases the respiratory distress. Intravenous injection of a 1 per cent. solution of methylene blue affords considerable relief, possibly because methylene blue is a powerful hydrogen acceptor, and thus liberates intramolecular oxygen in the tissues.

Report of an Incident

Towards the end of 1943 a ship docked at a port in the Persian Gulf. Part of the cargo was paranitraniline packed in wooden kegs, a number of which had been broken during the voyage, resulting in one of the ship's holds becoming coated with the yellow dye in the form of a fine powder.

During the night a shift of Persian dock labourers was engaged in unloading the unbroken kegs and cleaning out the hold. Cleaning was performed in the Oriental manner of squatting on the heels and sweeping with hard brooms. In consequence the air became heavily laden with paranitraniline, which was inhaled freely by the sweepers. Their skins and clothing also became covered with the dust. The shift ceased work at about 6 a.m. At mid-day seven of these labourers attended hospital, all presenting the same syndrome in varying degree, one of them being unconscious. They complained of intense headache, somnolence, weakness, and respiratory distress. Their lips, the tips of their ears, tongue, finger-tips, toes, and to a lesser extent, their faces and bodies, were mulberry coloured. Their clothing was stained with yellow powder. The pulse in most cases was regular, but rapid and of poor volume, the blood pressure was normal and the respiration rate was raised to about 26 per minute in all cases. Respiratory distress became most marked on slight effort. None of the patients vomited.

Three of the more severely affected were admitted to hospital, two conscious, and the third unconscious, though he was restless and irritable, and resented interference. The unconscious patient had his spleen enlarged to four finger breadths below the costal margin, and liver to two finger breadths with a hard and easily palpable edge. The upper limit of liver dullness was in the seventh intercostal space. The two conscious patients were admitted because of intense headache, extreme dyspnoea, and deep cyanosis.

A concentrated solution of methylene blue in

sterile distilled water was prepared and 10 ml. administered intravenously to each patient. The results were immediate. The unconscious patient vomited mucous material stained bright yellow by the dye, and regained consciousness sufficiently to ask for water. Five minutes after the injection the conscious patients volunteered the information that their headache had almost gone, and the relief of their respiratory difficulty was obvious. Nausea and a little retching occurred in both cases, but it is thought that if the injection had been given more slowly this might have been avoided.

In spite of the relief of subjective symptoms the cyanosis remained unchanged for about half an hour. It disappeared slowly, however, over a period of 12 hours, by the end of which time the two conscious patients had fully recovered.

The unconscious patient was less fortunate. An hour after the injection of methylene blue solution, which was given at about 4.30 p.m., he was answering simple questions. The following morning he was still conscious but had developed marked jaundice accompanied by pyrexia, tachycardia, albuminuria, and haematuria. The blood urea was 137 mg. per 100 ml. No malarial parasites were found in the peripheral blood, although he undoubtedly had a malarial hepatitis and splenitis. In spite of all efforts, he died at 2 a.m. on the morning of the third day, about 36 hours after admission to hospital or 50 hours after the commencement of his eight hours' exposure. Religious objections prevented the performance of a necropsy.

The ship was visited about three hours after these cases were first seen, and several of the European crew were found to be suffering mildly from the effects of paranitraniline absorption, notably the 'bosun, who had been supervising the cleaning operations from the hatch. None of the Europeans had been in the hold, but there had been a heavy cloud of the dust over the fore end of the ship. The Master was ignorant of the hazard involved, and no warning had been issued to him. The nature of the powder was only discovered after the ship's papers were inspected. The kegs were not marked in any way, and there was no other indication that their contents were poisonous. There had been similar cases at a port of call some days previously, when the same hold had been open and unbroken kegs were unloaded. The hold was subsequently washed out with hoses by men wearing canister-type masks, without untoward incident.

Discussion

Recorded cases of acute paranitraniline poisoning are few. Hamilton (1929) quotes a fatal case from a dye works at Hoechst, where a man had worked for five hours in paranitraniline dust. Motashaw (1945) reported two cases in Bombay, one of which was fatal. These men pushed a hand-cart containing a leaky cask of paranitraniline a distance of eight miles, and on the return journey one became ill. The other placed him on the cart, which was still covered with the powder. Subsequently, the second man began to weaken, so both lay down on the pavement to sleep, and were found in a comatose condition several hours later. The first case died. Two further cases are described by this author from the records of the King Edward Memorial Hospital, Bombay, covering the previous ten years.

The incident described here well illustrates the 'lag' period before the onset of symptoms. The men finished work at 6 a.m. but did not appear at the hospital till mid-day. The hot, moist conditions in the hold of a ship in the Persian Gulf probably assisted the absorption of the toxic substance. Mean minimal temperatures at the time of the year are usually between 65° and 90° F., while the clothes and skins of the patients, being heavily coated with the dye, assisted continued absorption even after work had ceased. It is stated that a moderate degree of aniline poisoning will recover in about 24 hours, but that recovery from paranitraniline poisoning takes longer. Two of Motashaw's cases took four days to recover. The two cases recorded here had completely recovered in 24 hours, but the 'bosun, who was not at first severely affected, still had headache and recognizable cyanosis at the end of the same period of time. Jaundice has never been described in any form of aniline poisoning, but it is probable that the malarial hepato-splenitis existing in the fatal case was transformed into a toxic jaundice by the added strain of the attempt to deal with the poison. Scheff (1940) has shown that the liver plays a part in the metabolism of both aniline and its probable degradation product, *p*-aminophenol. The basic cause of the incident was lack of appreciation of the risk. The Master had had no warning that he carried a potentially toxic substance and the containers were not labelled.

Summary

The occurrence of paranitraniline poisoning in Persian dock labourers handling the dye in powder form in an enclosed space is described.

The intravenous injection of a concentrated solution of methylene blue brought about rapid relief of symptoms in three patients to whom it was administered.

A malarial hepato-splenitis in one case was transformed into a toxic jaundice by the action of the poison, resulting in death.

Ignorance of the hazard on the part of those responsible for handling the dye was due to lack of information.

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THE OCCUPATIONAL HAZARD OF DDT SPRAYING

BY

I. GORDON

From Lagos, British West Africa

The ideal insecticide, like the ideal antiseptic, should have as one of its main properties a marked lethal effect on the organism attacked, and a minimal toxic effect on man or animals. Thus it is no accident that DDT and penicillin have assumed so prominent a place in hygiene and therapeutics respectively, for they both are strongly toxic to certain undesirable forms of life and relatively harmless to man. However, evidence as to the lack of toxicity to man of DDT, up to date, is almost entirely confined to laboratory experiments on animals, a few human volunteers, and personnel engaged in the manufacture of DDT. An excellent opportunity presented itself to me on arrival in West Africa to investigate the toxic action of DDT in the field on a team of twenty-seven whole-time African sprayers, civilians employed by the army. The duration of the experiment was six months, from May to October, 1945, when it had to be concluded as the labourers were discharged. During these six months 28 cwt. 78 lb. of crude DDT, dissolved in 7428 gallons of kerosene were expended, and on an average each man was responsible for the distribution of over one hundredweight of the crude powder dissolved in kerosene in the proportion of 6·8 oz. to the gallon. The percentage purity of the DDT varied considerably from sample to sample, some batches being as low as 52 per cent., others up to 61 per cent. Spraying was carried out on five days of each week, on all military accommodation in Lagos; nine of the men were also employed in spraying DDT on mosquito breeding grounds. The sixth day was spent in repairing apparatus and vehicle maintenance. The apparatus used was either the Mackenzie or the Four Oak's sprayer. At first complete instructions were issued for personal protection against possible absorption, the wearing of gloves, gas capes, and eye shields, and for provision of an anti-drip can on the Mackenzie sprayer to catch the large amount of spray that ran back from the nozzle on to the operator's hands. However, it was soon apparent that no amount of ordering would convince these African civilians of the necessity for precautions, and it may be accepted that after the first few weeks no precautions at all were taken. However, there was no great risk, as the men were kept under constant supervision. The only exception was that personnel actually engaged in mixing and dissolving the DDT had to wear elbow-length gloves. This could be insisted upon as the individuals concerned worked within visual distance of the orderly-room.

Considerable work has been done on the toxic properties to mammals of DDT (Cameron (1945); Buxton (1945)). DDT in powder form applied to the skin is harmless, but if dissolved in an organic solvent such as acetone or kerosene it can be absorbed through the skin and cause poisoning. DDT in aerosol or mist form in any strength likely to be used has been shown to be non-toxic to human beings. Ingestion of small amounts regularly will eventually cause poisoning in man, but apart from eating sprayed fruit or vegetables, or by accidental ingestion, this hazard is not likely to occur. Only three cases of poisoning in man have been reported. Wigglesworth (1945) described poisoning in a laboratory worker who carelessly handled DDT in acetone; Case (1945) reported evidence of absorption through the skin in two volunteers under certain specific experimental conditions; and Hill and Robinson (1945) reported the death of a 19 months old African child who drank one ounce of 5 per cent. DDT in kerosene. Early warning of intoxication may be the appearance of anorexia. Nervous manifestations, such as hyperexcitability, inco-ordination, muscular weakness, tremors, and convulsions predominate. Post mortem, the most obvious sign is degeneration of the liver. Cameron (1945) states that experience in animals has shown that features of value in assessing toxic absorption are a rapid decrease in weight, development of anaemia and leucocytosis, and perhaps a rise in blood calcium. Accordingly, in this work, these have been the signs looked for. There are no records of dermatitis from DDT, and sensitization is uncommon (Buxton, 1945).

Present Investigation

The spraying began in May, but this investigation did not commence until July. This meant that data were, unfortunately, not available concerning the subjects before spraying commenced. At the end of July, August, September, and October each subject was weighed and examined clinically. Blood calcium estimations, haemoglobin, white blood count, and differential were done on each

individual twice, one in August and again in October.

At the end of August, not only was the average weight of the sprayers less than 9 stone, but the average weight was going down, and the number who lost weight was seventeen as compared with eight

TABLE 1

The results of the weighings are as follows:

	July	August	September	October
J.E. ..	131	130	130	132
O.I. ..	120	120	120	122
U.E. ..	115	111	121	122
A.Y. ..	126	125	129	132
B.A. ..	118	115	111	119
P.I. ..	118	120	115	120
E.O. ..	136	138	140	142
O.O. ..	138	134	135	138
O.A. ..	118	115	112	115
O.A. ..	106	101	105	108
S.B. ..	115	116	117	120
W.O. ..	126	124	125	130
J.O. ..	150	149	152	155
D.E. ..	115	111	114	122
O.L. ..	120	120	120	121
O.O. ..	127	126	124	131
O.R. ..	160	159	164	162
J.O. ..	118	116	121	120
O.V. ..	117	113	105	105
A.A.B. ..	131	130	132	Sacked
S.A. ..	132	130	131	138
D.A. ..	111	115	129	128
M.M. ..	133	131	132	132
T.D. ..	116	112	115	115
J.O. ..	126	126	131	130
A.S. ..	134	136	135	142
B.A.B. ..	130	130	134	135

TABLE 2

	No.	Average Weight	Gains	Losses	No change
End July ..	27	125.4	—	—	—
End Aug...	25	125·0	4	17	4
End Sept...	27	125·9	17	5	3
End Oct. ..	27	127·9	19	4	3

stationary or gaining. It was then that the absence of a control group was felt. Thirty grass-cutters were chosen and weighed monthly from the end of August on. Their weights were as shown in Tables 3 and 4.

It will be seen from this that at the end of August the controls weighed slightly less than the DDT workers, and in the subsequent two months the course in the two groups was much the same, so the poor weights were unlikely to be due to DDT. Thirteen soldiers were then weighed and the average weight was 141·3 lb., 24 lb. more than the average of the

TABLE 3

	August	September	October
B.O.	115	119	118
S.E.X.	131	133	130
P.O.	130	134	128
R.I.	135	140	136
R.E.	112	120	116
J.O.	111	120	120
J.A.	132	139	138
A.N.	92	102	—
S.A.	130	128	130
E.O.	110	113	—
J.I.	116	120	116
U.J.	122	131	130
O.S.	120	125	120
U.T.	130	130	130
J.O.	112	115	130
E.J.	122	129	129
O.S.	121	124	126
P.A.	111	118	—
E.S.	110	113	114
I.J.	116	115	126
U.M.	112	115	117
S.O.	124	128	128
A.A.	118	111	—
A.J.	141	147	148
M.E.	126	130	138
M.O.	104	105	101
A.U.	111	114	116
D.I.	111	114	104
J.N.	132	131	128
D.U.	116	116	118

TABLE 4

	No.	Average Weight	Gains	Losses	No change
End Aug...	30	119·1	—	—	—
End Sept...	30	122·6	24	4	2
End Oct. ..	26	124·4	10	12	4

civilians. The combined average weight of the thirty civilians and thirteen soldiers was 126 lb.

Thus the difference in weight between the civilians

TABLE 5

	Civilians	Soldiers	Total
Above Av. Wt. ..	9	10	19
Below Av. Wt. ..	21	3	24
Total	30	13	43

$x^2 = 6{\cdot}3 \quad P > {\cdot}01 < {\cdot}02$

and soldiers is significant. As there are a number of tribes involved, difference in weight might have been due to difference in tribe. All the civilian controls were Ibos, the average weight being 119·1 lb. Of nine Ibo soldiers the average weight was 143·4 lb., a similar result to civilians and soldiers generally. As the difference was not due to tribe,

TABLE 6

WHITE BLOOD COUNTS AND DIFFERENTIAL COUNTS

(First column in each case refers to August, second to October.)

	W.B.C.		In. or dec.	Poly.		Lymph.		Mono.		Eosin.		Baso.	
J.E.	5000	10600	+	60	58	30	33	2	4	7	5	1	0
O.I.	6800	9000	+	66	72	31	21	0	4	3	3	0	0
U.E.	8000	9800	+	58	66	28	27	4	3	10	4	0	0
A.Y.	8000	9600	+	45	58	52	38	1	2	2	2	0	0
B.A.	4000	9800	+	65	70	28	22	1	5	6	3	0	0
P.I.	8500	10000	+	66	70	30	27	1	3	3	0	0	0
E.O.	5800	6800	+	61	62	34	29	2	2	3	6	0	1
O.O.	5000	11200	+	70	66	24	28	2	3	3	3	1	0
O.A.	4000	11000	+	75	72	20	20	1	6	4	2	0	0
O.A.	3900	9600	+	67	66	24	29	4	2	5	2	0	1
S.B.	4900	6800	+	66	66	27	30	1	2	6	2	0	0
W.O.	8000	9200	+	61	60	34	32	1	4	4	4	0	0
J.O.	4800	10000	+	70	59	25	33	1	3	4	4	0	1
D.E.	5000	7800	+	59	63	31	27	2	5	8	5	0	0
O.L.	6000	9600	+	62	63	32	28	3	5	3	4	0	0
O.O.	8000	8800	+	67	63	25	34	2	3	5	0	1	0
O.R.	5500	7800	+	63	52	30	44	1	4	6	2	0	0
J.O.	7700	8000	+	67	78	27	12	3	6	3	3	0	1
O.V.	5100	7600	+	64	66	32	28	1	2	3	2	0	2
A.A.B. ..	6800	—		60	—	35	—	1	—	4	—	0	—
S.A.	9000	7000	−	69	68	26	24	1	6	4	1	0	1
D.A.	8600	6800	+	66	68	27	26	3	5	2	3	2	1
M.M. ..	7500	8800	+	65	72	27	19	6	6	2	3	0	0
T.D.	5300	7200	+	65	66	26	27	3	3	5	4	1	0
J.O.	9000	—		74	—	23	—	3	—	0	—	0	—
A.S.	7000	—		65	—	30	—	2	—	3	—	0	—
B.A.B. ..	6400	—		58	—	32	—	4	—	6	—	0	—

and as all the people involved lived in Lagos, it was probably due to malnutrition and, perhaps, a certain pre-enlistment selection, a supposition fully supported by economic conditions in Lagos at that time. The civilians were paid 2*s.* a day, and food was scarce and controlled. Political tension ran high, and in late July and early August there was a strike of all transport workers, making the food situation even more difficult. After the strike conditions improved, and this was reflected in the slight gains in weight of the civilians in September and October. The final conclusion is, therefore, that there was no significant change in weight that could be attributed to DDT.

Leucocyte Counts

The individual counts show little of significance. However, it may be noted that of twenty-three white blood counts, twenty-one showed an increase between August and October. The counts were made in the morning. Unfortunately there was not a control series, so there can be no certainty that this was due to DDT.

Haemoglobin and Blood Calcium

In August haemoglobin and blood calcium estimations were done on the DDT sprayers. For the haemoglobin the Sahli method was used. The main feature is that the October haemoglobins and blood calciums both lie within normal limits. A certain minor erratic distribution of some results is due to the varying technical abilities of the laboratory assistants.

TABLE 7

	Haemoglobin		+ or −	Blood Calcium		+ or −
	August	October		August	October	
J.E. ..	14·5	14·1	−	10·0	9·5	−
O.I. ..	11·3	14·1	+	9·5	9·1	+
U.E. ..	14·0	13·2	−	8·0	9·5	+
A.Y. ..	12·5	14·1	+	9·5	10·0	+
B.A. ..	12·6	13·8	+	9·3	9·2	−
P.I. ..	13·5	13·5		9·0	10·6	+
E.O. ..	14·5	13·1	−	10·0	9·5	−
O.O. ..	15·0	13·2	−	9·5	9·8	+
O.A. ..	12·0	13·1	+	8·5	9·6	+
O.A. ..	12·4	13·8	+	10·0	10·0	
S.B. ..	14·0	13·8	−	8·5	9·6	+
W.O.	12·2	13·5	+	10·0	10·4	+
J.O. ..	13·2	13·5	+	9·5	10·4	+
D.E. ..	10·7	13·2	+	9·0	10·0	+
O.L. ..	13·5	13·8	+	9·8	9·0	−
O.O. ..	14·0	13·5	−	10·0	10·6	+
O.R. ..	14·4	13·2	−	9·8	10·5	+
J.O. ..	15·0	13·8	−	10·5	9·3	−
O.V. ..	14·6	13·8	−	9·5	10·0	+
A.A.B.	13·0	—		9·5	—	
S.A. ..	12·3	13·8	+	9·2	9·8	+
D.A. ..	13·5	13·2	−	10·0	10·3	+
M.M.	14·0	13·8	−	10·8	9·3	−
T.D. ..	14·5	13·5	−	9·5	10·0	+
J.O. ..	13·4	—		10·5	—	
A.S. ..	13·8	—		11·0	—	
B.A.B.	16·8	—		10·5	—	

Skin Rashes

Apart from the rashes to be mentioned, nothing of note was found at the monthly inspections. Some acquaintance with the literature, experience in Europe with anti-louse powder containing DDT and liquid spraying with a kerosene solution would convince anyone that no trouble was to be expected with skin irritation. However, immediately on arrival in West Africa a different story was told me by the N.C.Os., who stated that skin trouble was prevalent, especially when DDT spraying first started. Examination of the sprayers, however, showed that whatever might have been the situation in May they were free from trouble in July, with the exception of the mixer, A.A.B. It was decided to investigate the situation by means of patch tests.

The first week in September 27 DDT workers were tested, and shortly afterwards 30 controls (grass-cutters). A piece of lint about 1 in. square was lightly damped with the test solution, 5 per cent. DDT in kerosene, and fixed to the right arm with plaster. Kerosene only as a control was placed on the left arm. The patch was applied at 4.30 p.m. and read at 8.30 a.m. the next day. The result among the non-DDT contacts was as follows:—

TABLE 8

No.	Test Arm	Control
12	−	−
2	+	+
13	+	−
1	++	−
2	+++	+

Four showed a small reaction with kerosene, all four of whom also showed a similar or larger reaction with the DDT in kerosene on the other arm. Fourteen showed a reaction to the DDT in kerosene, but not to the kerosene alone. The reaction was vesicular, the smallest rash consisting of a few scattered vesicles the size of a pinhead, and the largest a blister the size of the patch, with oedema. These 30 controls were next similarly tested with a 5 per cent. solution of DDT in dimethylphthallate (DMP). None showed any evidence of a rash whatsoever. It is well established in Europe by the delousing of millions that DDT powder is non-irritant, as shown here DDT in DMP is non-irritant, kerosene (for this experimental contact period) was slightly irritant, but 5 per cent. DDT in kerosene considerably irritant. This is true of Europeans (of whom 6 were tested) as well as Africans. The conclusion seemed to be that DDT, whilst innocuous on its own, considerably enhances the vesicant action of kerosene. This applies to the controls, who had not been in contact with DDT. With the 27 DDT contacts the results were quite different, in early September only 3 showed a moderate reaction to the DDT in kerosene, and none to kerosene alone, one of these three was the mixer, but by the end of October, out of 26 remaining none showed any reaction to DDT in kerosene or the control. This difference is, of course, highly

TABLE 9

	DDT Workers	Controls	Total
Positive to either test or control ..	0	18	18
Negative to both test and control	26	12	38
Total	26	30	56

$x^2 = 20{\cdot}3$

significant. A.A.B., the mixer, was noted to have a rash, papulosquamous in type, on arms, front of abdomen and thighs. He was also positive to the patch test. He was taken off mixing and the rash improved. However, the rash could not be followed up for a long period as, owing to drunkenness, he had to be discharged. A soldier was put in his place, and also developed a rash, but he in his turn could not be investigated as he was sent to prison. The third soldier who took on mixing likewise got a rash, which, however, soon cleared up.

It seems, therefore, that sensitivity to DDT in kerosene disappears with continual contact, and that the mixers are far more likely to get a rash than the sprayers.

This was well illustrated early in December when soldiers took over spraying from the discharged civilians. Complaints of rashes among the sprayers were soon made, 5 out of a total of 12 had some trouble, as compared with none of the recently-discharged civilians. Reference in literature to irritation seems confined to the description by Nelson and his colleagues (1945) in experimental animals of a mild dermatitis with hyperkeratosis and very little cellular infiltration. In more severe cases there was a slight focal epidermal necrosis. Macdonald, in a discussion after presentation of a paper by Buxton (1945) records dermatitis in a sprayer, and states there was no evidence to differentiate it from a kerosene rash; Cameron (1945) describes severe inflammation of the skin in rabbits who had been subjected to cutaneous applications of DDT in kerosene. He suggested that the kerosene alone might be responsible for this.

Discussion

That the vehicle in which DDT is incorporated may cause more trouble than the DDT is apparent with kerosene. Anti-louse powder is made up with china-clay, and if powdering is performed indoors, either by ignorance of the correct technique or because of weather that makes it impossible to carry on out of doors, the room becomes full of a cloud of dust. Sore eyes, nose and throat are soon complained of, and it becomes impossible to

continue working in the atmosphere without wearing a mask, all the symptoms undoubtedly due to the diluent. Manufacturers should keep in mind the fact that the vehicle may cause more complaints than the active substance, which in practice has never caused any trouble.

It would not be out of place here to suggest that kerosene be used no longer than is absolutely necessary as a solvent for DDT. It has been used hitherto because it is in good supply and cheap. As kerosene has been available locally, DDT could be shipped in powder form and thereby save considerable shipping space. The disadvantages are that kerosene has an objectionable smell, may stain walls, ruin rubber and fabrics, and be irritant to the skin; and it is immiscible with water. DDT should be provided in an emulsion form when a liquid spray is required as soon as is commercially practicable.

Summary

1. Blood examinations, regular weighings, and skin inspections have been carried out on 27 African whole-time DDT sprayers. Although these men were probably undernourished and took no precautions against skin absorption, no signs or symptoms of DDT poisoning were found. Slight increases in the leucocyte count were noted.

2. When DDT was used in kerosene, rashes were common when sprayers first started on the work. After some months, however, they became desensitized. By means of patch tests it was shown that the irritation is primarily due to the kerosene, and made considerably worse by the DDT dissolved in it. DDT alone is innocuous. A plea is made for the discontinuance of kerosene as a solvent for DDT.

Acknowledgements

My thanks are due to Brigadier H. B. F. Dixon, D.D.M.S., West Africa Command, for permission to publish this article, and to Cpl. Bland, R.A.M.C., for the bulk of the blood examinations. When it is considered that this work had to be carried out in spite of absences of many weeks at a time whilst supervising a unit of which one detachment is as far from the other as London is from Moscow, the difficulty in producing a perfectly controlled experiment may be realized.

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OLEOGRANULOMA

THE LATE EFFECTS OF ACCIDENTAL INJECTION OF MINERAL OIL UNDER PRESSURE

BY

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Accidental introduction into the tissues of mineral oil under pressure is an occupational hazard which has come into some prominence in recent years. The picture presented by victims of this accident in the early stages has been stressed in all the published reports. The aim of this paper is to draw attention to the disastrous late effects that may follow such an accident. These have been predicted by a number of writers, largely by analogy with certain comparable conditions; but I have not been able to find records of any late cases in the literature.

The characteristic early manifestation of this type of accident is the grease-gun injury to the hand (see Brooke and Rooke 1939, Smith 1939, Byrne 1944). This is associated with the accidental injection into the subcutaneous tissues of the fingers of lubricating grease delivered under high pressure from the nozzle of the grease-gun now commonly used in garages. A similar type of injury has been reported in Diesel engine operators (see Rees 1937, Dial 1938, de Lacerda Filho 1941, Hughes 1941). Mason and Queen (1941) described an intermediate stage following grease-gun injuries, in which nodular tumours developed at the site of injection of the mineral oil and persisted with apparently little change for considerable periods. They showed that histologically the tumours were essentially the result of an intense productive fibrosis around a nidus of mineral oil in the tissues. The term 'oleogranuloma' more aptly reflects, therefore, the pathological process involved, and is used here in preference to the term 'oleoma' used by the above authors and other writers.

Mason and Queen stressed the importance of adequate surgical removal of the mineral oil from the tissues, predicting that serious late results would otherwise follow. This view was based on the work of Hesse which will be mentioned below; but it is supported by a number of parallels in the literature, which may be briefly summarized:—

1. In 1899 it was suggested that paraffin of high melting point should be used as a means of correcting disfigurements of the face or other parts of the body. The results at first appeared satisfactory, but it was later found that in a relatively small proportion of cases the end-results were disastrous owing to the formation of hard subcutaneous nodules which developed at the sites of injection and spread into the surrounding tissues. Chronic ulceration and scarring followed, and the only satisfactory treatment of the affected tissues was complete excision followed by skin grafting.

2. During the Great War of 1914–1918, the injection of mineral oil into the lower limbs was practised in White Russia as a way of evading military service. The rationale of this was that the oil was known to produce a factitious tumour, and according to an old Russian law any man unable to wear the regulation high field boot was exempt from military service. Hesse, of Leningrad, described 183 such cases in 1925. The majority of these presented with bony-hard tumours, usually around the knee joint; some had, in addition, overlying ulceration and scarring. Eleven years later, in 1936, Hesse's assistant, Vinogradov, published a paper describing some late results in Hesse's cases, and this paper is the only one in the literature which gives a complete natural history of oleogranuloma from the early stages to the development of late sequelae. In advanced cases, the nodular tumours caused lymphatic obstruction and elephantiasis; multiple ulcers developed following trivial injuries and healed slowly with abundant scar tissue, and the final stages showed chronic deep ulceration occasionally with malignant change. A proximal spread of the tumour masses, sometimes with lymphnode enlargement, was also noted, and was correctly attributed to spread of the foreign oil particles along lymph channels. Amputation was the only satisfactory treatment.

3. Oleogranuloma of the upper limbs was described in the years following the influenza pandemic of 1918 as a result of multiple injections of camphorated oil, the vehicle being liquid paraffin. A typical case was that described by Mook and Wander (1919), which showed the same 'concrete-like' infiltration, the elephantiasis, and the proximal spread of nodules along lymphatic channels which the Russian authors later described.

4. Another considerable group comprises the oil strictures of the rectum, due to injections of phenol in oil in the treatment of haemorrhoids. Rosser published a series of 26 cases in 1931, with symptoms ranging from rectal neuralgia to complete obstruction, and proved experimentally that the mineral oil and not the phenol was responsible (see also Jackman 1940).

5. Finally, one may mention lipoid pneumonia,

the adult type of which is due usually to long-continued instillation of mineral oil sprays into the upper respiratory tract. Clinically these cases simulate pulmonary neoplasm, and the histological picture is essentially the same as is seen in the other oleogranulomas (see Ikeda 1937). There is, therefore, abundant evidence of the disastrous late results which follow the injection of mineral oil into the tissues; and it is clear that the only way to avoid these late sequelae is adequate surgical removal of the irritant oil as early as possible.

Case Report

The following case report is a distressing commentary on the need for radical treatment of such cases at an early stage:—

Case History. Male, aged 47. Occupation: Engineer's erector. A well-built man with no previous medical history. Six years ago, while working on high scaffolding, he slipped and fell about six feet on to a pipe containing a heavy lubricating oil under pressure. The end of this pipe penetrated a few inches through the right side of his scrotum and into his perineum, and then snapped across, so that an unknown volume of oil was injected under pressure into his ischio-rectal tissues. He extracted the broken pipe, and being anxious to finish the task on which he was engaged, continued with his work. The wound healed within a few weeks without any treatment.

Nine months later, i.e. about five years ago, he was admitted to a hospital in Derbyshire with a large acute perineal abscess, which was drained. He spent four months as an inpatient in this hospital before his wound healed.

Two and a half years ago, in October 1943, he was first admitted to the Radcliffe Infirmary, Oxford, under Mr. Whitelocke. He presented as a cellulitis, with temperature 100° F, pulse 80; and showed a large, hard, painful swelling which extended forward from the perineal region to the front of the right pubic ramus, with an extension into the right side of his scrotum. He was treated with sulphonamides, and the swelling subsided after three weeks. During this time he had acute retention of urine for a few days. This responded rapidly to treatment with an indwelling catheter, but since then he has lost the normal stimulus to micturition, his first indication of vesical distension now being hypogastric pain.

When his acute cellulitis subsided, it was found that he had a hard mass extending upwards along the right side of the rectum as far as a finger could reach, and hard nodules in the skin of the perineum, one in the scrotum and one in the anterior rectus sheath. Biopsy was performed on the ischio-rectal tissues, and the histological diagnosis of oleogranuloma was made. The biopsy wound healed very slowly and a scrotal abscess developed two months after the operation and was incised. Cultures yielded Staph. aureus and β-haemolytic streptococci. Further outpatient treatment lasting five months preceded his discharge in May, 1944.

During the following four months he had increasing pain and tenderness in the scrotal nodule, which was gradually becoming larger. He also began to suffer from severe hypogastric pain, especially at night.

In August, 1944, he was readmitted to the Radcliffe Infirmary and the scrotal nodule was excised. Six weeks later he was sent to a convalescent home with his operation wound still unhealed. Here his abdominal pain became more severe and on October 22nd he was readmitted to the Radcliffe Infirmary as a possible acute abdominal emergency, with extremely severe hypogastric pain radiating into both loins. This pain gradually responded to morphine, and he returned to the convalescent home. In December, 1944, he was discharged to his home in the North Midlands at his own request. Follow-up inquiries supplement the history as follows:—

16.5.45. Admitted to his local hospital for incision of an ischio-rectal abscess. He was discharged a week later with his wound still draining.

14.6.45. Readmitted with orchitis and scrotal cellulitis. He was treated with sulphonamides. Ten days later a discharge of pus from his scrotum started, and this continued for four weeks. He was finally discharged on 7.8.45.

7.9.45. In a personal letter, the patient said he was still suffering from abdominal pain and that his scrotal wound was not yet healed.

21.3.46. Patient still unable to resume work and complaining of a hard scrotal swelling and a discharging wound in his right groin.

Finally, it may be mentioned that the engineering firm by which the patient was employed has refused responsibility for compensation on the grounds that at the time of the accident the patient was working single-handed. This contravened one of their regulations which directed that no employee should work on a scaffolding without having an assistant to help him.

The macroscopic and microscopical appearances of the two specimens from this patient are very similar, and correspond very closely to the findings in other types of oleogranuloma. Fig. 1 is a photograph of the scrotal nodule which was excised in September, 1944. This nodule measured 4×4×2 cm. and was firm in

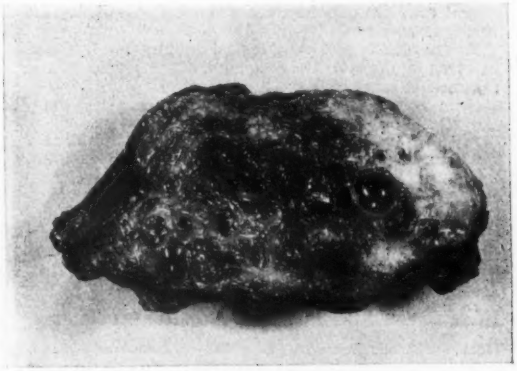

FIG. 1.—Section of scrotal nodule showing oil-containing cavities in a dense mass of fibrous tissue. Approximately natural size.

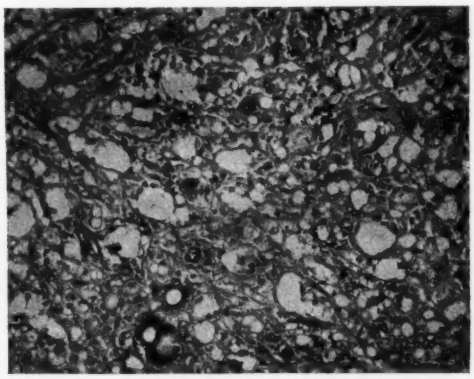

FIG. 2.—Granulomatous tissue with empty spaces representing encysted oil (×110).

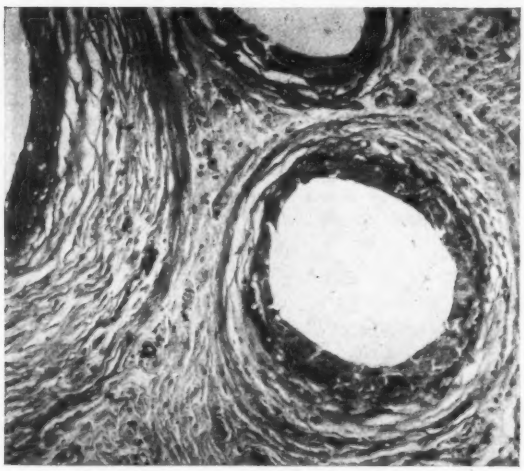

FIG. 3.—Larger space with layer of flattened histiocytes inside the fibrous tissue capsule (×110).

consistency. The cut surface was yellow and greyish-yellow in colour and was studded with numerous cavities containing an amber-coloured oily fluid.

Microscopically, one sees what is essentially a marked foreign-body reaction with a characteristic picture of active granulation tissue enclosing the foreign oil in cysts of very varying sizes, which are represented by empty spaces in ordinary H. & E. preparations (fig. 2). The larger spaces are ringed by a fairly dense fibrous tissue capsule, inside which is a layer of flattened histiocytes (fig. 3). The latter, in sections stained with Sudan, are seen to contain fat globules. There are numerous histiocytes scattered through the fibrous tissue of the nodule, and a few polymorphonuclear leucocytes, and occasional foreign-body giant cells. Sections stained for fat show large numbers of globules staining orange-red with Sudan and occupying the large and small spaces seen in paraffin sections. Under the fluorescence microscope, the globules, examined in unstained frozen sections, show a vivid blue fluorescence.

Discussion

The course of the chronic illness manifested by this patient is very similar to that followed by Hesse's cases. Owing to the site of the lesion the mechanical results of lymphatic obstruction are not evident, but the recurrent infections are an index of the devascularization and diminished resistance caused by the intense productive fibrosis occurring in this disease. It is possible that the severe abdominal pains are associated with upward spread of the granulomatous process along lymphatic channels, as was seen in Hesse's patients.

The progressive course of oleogranuloma suggests that the pathological process is more than a simple foreign-body reaction on the part of the tissues to the presence of an inert foreign substance. One might postulate some chemical irritant effect comparable to that which plays an important part in the pathogenesis of silicosis. There is, in fact, some evidence that this may be the case. Workers handling the heavy fraction obtained on distillation of crude oils are subject to severe skin reactions ranging from the results of mechanical blockage of sweat-ducts and hair-follicles to a definite epithelial proliferation in the form of warts which occasionally become malignant. These lesions are probably due to impurities which for technical reasons are difficult to remove; they do not occur in workers who handle purified solid paraffins. Another fact which supports the idea of a chemical irritant effect is the relatively low incidence of oleogranuloma in cases where paraffin was used for plastic repair. If the response to the injection of paraffin were a simple foreign-body reaction, one would have expected the development of oleogranuloma and its effects in at least the majority of such cases. Some writers have attempted to explain the matter on a basis of allergy, but it seems more probable that certain batches of paraffin contain impurities which stimulate excessive fibrosis in patients into whom they are injected.

Summary

1. Previous reports concerning the late effects of parenterally introduced mineral oil on the tissues are briefly reviewed.

2. The clinical and pathological findings in a late case of oleogranuloma of the perineum, caused by accidental injection of lubricating oil, are described.

Acknowledgement

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CALCIUM NECROSIS OF THE SKIN

BY

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A method of dust suppression recently introduced into mining depends upon the hygroscopic properties of calcium chloride, a 40 per cent. aqueous solution, to which is added 2–4 per cent. of a wetting agent (' Lissopol N ' I.C.I.), being used for spraying the roadways of mines. Contact with the solution was presumed to be innocuous but the following case showed that this was not true.

Case Record

J. C. W., while engaged in spraying in a South Wales coal mine, allowed some of the solution to contaminate his skin. Immediately after exposure, which lasted 8 hours, he experienced burning pain accompanied by the formation of ' wheals ' on the upper eyelids, forearms, and thighs. Similar effects were noticed on his hands and legs, where abrasions occurred during the work, those on the legs being due to friction from the tops of his rubber boots. Eleven days later the lesions on the legs and hands broke down. When first examined 16 days after exposure there were large sloughing ulcers with raised, dark red edges running transversely across the upper parts of both legs and smaller round ones on the backs of both hands, with crusts which, when removed, left deep pits. The lesions on the eyelids, forearms, and thighs were not ulcerated but consisted of pearly yellow, raised spots measuring 1–2 mm., some of them round and others shaped like splashes.

These lesions have been observed for over 5 months. The ulcers were indolent, and epithelialization was not completed until after 13 weeks, but from the seventh week onwards those on the legs were outlined by narrow zones of yellow discolouration of the deeper tissues. The appearance of the non-ulcerated lesions did not change for the first 4 months, but they finally disappeared.

Another man who was engaged in the same work noticed burning and redness at the sites of contact immediately after exposure. He alleged that he then washed very thoroughly with soap and that the effects disappeared in about a week.

Histology

Biopsies were taken from lesions of the forearm, hand and leg 18 days after exposure. The essential features were shown best by one of the non-ulcerated lesions from the right forearm. This exhibited a sharply circumscribed area of coagulative necrosis of the dermis, the necrotic collagen fibres being heavily impregnated with calcium salt, while the overlying epidermis was normal, apart from some loss of cellular definition. Here no inflammatory reaction could be seen (fig. 1), but in a severely ulcerated lesion from the right leg the picture was modified by inflammation. There the calcium-impregnated tissue was less conspicuous and showed a granular fragmentation and cellular infiltration. Sweat gland epithelium in the affected area was damaged in some parts but escaped in others. A small ulcer from the right hand had an intermediate appearance, there being less inflammation and more calcium necrosis.

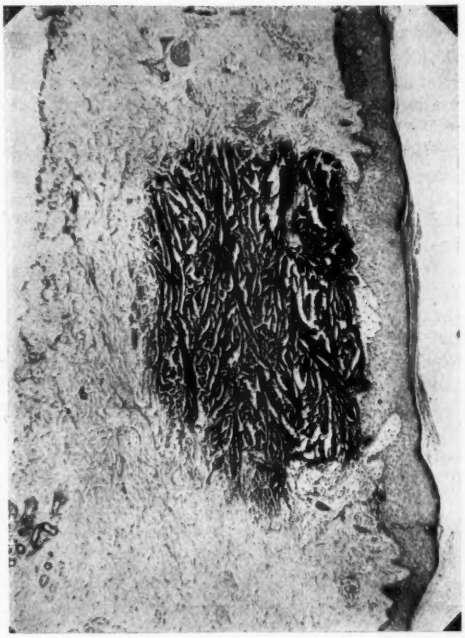

FIG. 1.—Non-ulcerated lesion, right forearm. Stained von Kossa, haematoxylin and eosin (×40).

Discussion

The development of two types of lesion apparently depended upon whether the skin was injured or not at the time of contact with the solution. In the absence of injury, calcium infiltration with necrosis of the dermis alone developed, but where there were abrasions, inflammation and ulceration followed. The progress of the severely ulcerated lesions on the legs also indicated that complete resolution only occurred where the inflammatory reaction was sufficiently intense to effect disintegration and discharge of the dead tissue. In the centre of the ulcers the reaction was marked, and healing, though rather delayed, was satisfactory. At their edges, where it was less intense, yellow zones remained; and

these, judging from the non-ulcerated lesions, represented persistent subepidermal deposits of necrotic material which in time may be removed without surgical intervention. It was significant that in both types of lesion healing took place most readily at the biopsy sites where the necrotic tissue was removed, showing that persistence of the latter was responsible for the delayed or incomplete resolution.

The fact that the epithelium over the non-ulcerated lesion was relatively normal (had the epithelium been regenerated one would not expect it to possess rete pegs) suggested that strong calcium chloride solution by itself can penetrate the epidermis and leave only minimal histological evidence of damage.

Lesions resembling those on the hands of this case were described by Oppenheim (1935) in an individual who, during the course of work as an ice-cream maker, dropped concentrated calcium chloride solution on to the insteps. Histological examination could not be made, so instead Oppenheim investigated the action of calcium chloride on the rabbit's skin. Intracutaneous injection of 0·1 c.cm. of a 10 per cent. solution evoked, after 3 weeks, lesions somewhat similar to the non-ulcerated ones observed in the present case; they differed in that an inflammatory reaction also developed. Scarification followed by the application of the solid salt induced a response comparable to the ulcerative type of human lesion. Calcium chloride was used specifically as a necrosing agent in the experimental study of silico-tuberculosis (Kettle, 1924) and of tetanus (Russell, 1927), but naturally neither of these lesions bore any histological resemblance to the human ones.

The part played by the wetting agent in the production of the lesions was uncertain. Its reputed harmlessness to normal skin when applied in a pure state suggested that a direct influence was negligible, but it may have facilitated penetration of the skin by the calcium chloride.

Summary and Conclusions

1. A case is described in which multiple cutaneous necroses developed as a result of contact with 40 per cent. calcium chloride solution.

2. The characteristic lesion was a non-ulcerated focal necrosis of the dermis without evidence of any kind of inflammatory reaction.

3. The lesions proved to be indolent and their delayed or incomplete resolution is attributed to the persistence of calcium-necrosed tissue. Where there was also inflammatory ulceration healing was assisted.

4. The use of such a solution for industrial purposes demands adequate protection of the skin and especially of the eyes.

5. Thorough and immediate washing, using plenty of soap, may mitigate the effects of contamination.

Acknowledgements

I am indebted to the patient for his co-operation and to Professor J. B. Duguid for criticism and advice. Mr. A. S. Aldis performed the biopsies and Mr. G. R. Armstrong rendered technical assistance.

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BOOK REVIEWS

MEDICINE IN INDUSTRY

By Bernhard J. Stern

The Commonwealth Fund. Geoffrey Cumberlege; Oxford University Press. 1946. Pp. 209. Price 8*s.* 6*d.*

The New York Academy of Medicine's Committee on Medicine and the Changing Order has selected titles and authors for a series of monographs. It is to this series that the present work belongs, and its author occupies a chair in Sociology. An American writing about American problems, he is careful to set against the advances that have been made the inadequacies that are apparent in the scheme of things, and to point out directions in which work needs to be done. An early chapter surveys the history of legislation on occupational diseases, draws the parallel between English and American laws, and discusses the complications of transference of responsibility from State to Federal authority. In a chapter on the incidence of industrial injuries and poisoning, accident rates are dealt with in relation to different industries; the discussion is supported by an abundance of statistics, many of which have obvious limitations, the statistics sometimes perhaps overburdening the text. The handicapped worker's present position is examined, and also the means available for his rehabilitation and placing in suitable work: it is interesting to note, bearing in mind the formation of our own Register of Disabled Persons, that the number of people with some disability in the U.S.A. is estimated at 24 million. Too many workers appear to be rejected at pre-employment examinations for inadequate reasons. A discussion of the preventive measures and the provision of adequate medical supervision in factories leaves the impression that all is not well, for there, as in this country, factory medical services are inadequate, and small plants very often do without the services of a doctor at all; strenuous efforts are being made, however, and it may be that American thought in the matter is in advance of our own. But the author points out that, even when there exists such good machinery as the Industrial Hygiene Division of the U.S. Public Health Service to investigate toxicological and other problems, the interest and co-operation shown by managements of industry is much below what it could be. This is an attractively produced volume with an extensive bibliography of the general aspects of the subject at the end of each section; it makes no pretence to be a work of reference, but there is plenty of food for thought in its pages, and as a sociological document it is admirable. One will wish to read it again in years to come when the Changing Order has had its chance to right the wrongs.

J. N. A.

N.A.P.T. HANDBOOK OF TUBERCULOUS ACTIVITIES

Edited by Harley Williams, M.D. 12th edition.

Pp. 275. 7*s.* 6*d.*

It is useful to have information on all tuberculous clinics, dispensaries, and sanatoria collected together as a directory. The handbook aims at producing a complete list of the facilities available for the treatment and care of a tuberculous patient—and it succeeds. It must have been difficult to obtain personnel to collect and correct the data; nevertheless, a book of this nature ought to be accurate. It contains many minor mistakes, especially in the spelling of people's names and in their descriptions. It is hoped that in the next edition this will be corrected, since this valuable directory deserves every success. As it states, to know the extent of the disease is one thing, to be aware of the facilities at present provided to conquer it is another. We hope that even the blanks and deficiencies revealed in these pages may provide a stimulus to further activity, and thus encourage opportunities for bringing modern methods and treatment within the reach of those who need them.

K. M. A. P.

JOB PLACEMENT OF THE PHYSICALLY HANDICAPPED

By Clark D. Bridges

McGraw Hill Book Company, Inc., New York. 1946. Pp. 329. 17*s.* 6*d.*

Here is a reference book written by a layman to help other laymen, employers, personnel managers, and all who may have to deal with the disabled. It will, too, provide a stimulus to factory medical officers who like to see their disabled people satisfactorily placed in work. The book should not escape the notice of any who practise in an industrial area, for besides providing for the non-medical reader a balanced, accurate, and reasonably simple summary of the commoner maladies and the disabilities they produce, it also analyses the physical requirements of industrial occupations in general in a way that is helpful to those whose practice does not often lead them into places where men do their daily work. The assessment of a man, and of the job he is to be asked to do, are both equally necessary for satisfactory placement; and after some pages in which the employability of disabled persons is emphasized, the author's attention turns to accurate assessment of jobs. A complicated specimen job-analysis form is shown, and its 155 sections are then discussed, with diverting sketches. The detail within the form is considerable, and it is doubtful if such a document would be useful except for someone whose whole time was given to job analysis. A complementary form demonstrates how a comprehensive medical examination for job placement is conducted and could be recorded; on a third form an attempt is made to simplify the doctor's recommendations to the personnel department about a disabled man. The largest section of the book describes specific disabilities caused by common illnesses or impairments of the special senses. These are arranged according to systems of the body, and in general are admirably described. Some of the medical terminology familiar enough to the doctor might prove difficult to the layman, for whom the descriptions are clearly intended, and it is a little difficult to see what advantage the latter can obtain from such highly technical information as the list of the signs which lead us to a diagnosis of quiescence, arrest, or apparent arrest, of a tuberculous lesion. A fuller explanation of peptic ulceration, and the sufferer's outlook and dietetic needs, might have taken its place. It is surprising, but welcome, to come upon detailed lists of chemical agents and the toxic manifestations to be expected from them, lists of the causes of toxic anaemia, and the like; but these might be of more value if they were shortened to include only the commoner causes. The extensive appendices include sections on evaluation of the severely handicapped; a list of institutions interested in industrial health problems in the United States, and of voluntary organizations for the rehabilitation of the handicapped, neither of which can assist us over here; and information on the maximum allowable concentrations of industrial air contaminants. The book is attractively produced, fulfils a very present need, and can be confidently recommended to a wider public than that for which it was intended.

J. N. A.

BRITISH AND FOREIGN OFFICIAL PUBLICATIONS

THE PRESIDENT'S NATIONAL LABOR CONFERENCE

(U.S. Department of Labor, Division of Labor Standards (1946). Bulletin No. 77.)

This publication contains the minutes of a conference held in 1945, the Summary Report to the President, and proposed representation procedure and agenda for the conference. The latter consisted of examination of the major causes of industrial strife and the methods of reducing them, including the principle of collective bargaining in matters of wages, hours, and working conditions, the acceptance by the workers of the right of the management to direct the enterprise, and the use of existing machinery, such as National and State Labor Relations Act, and negotiation between employer and Union. President Truman's opening address, on the aims of the conference and the urgent need for minimizing industrial disputes, is also included. Committee reports adopted by the conference deal in detail with collective agreements and the strengthening of the Conciliation Service of the U.S. Department of Labor and the reorganization of its Division of Arbitration. There are statements by representatives of managements and workers on collective bargaining, management's right to manage, and representation and jurisdictional subjects. There are reports of the Public Hearings Committee, in the fourteen hearings of which the interests of independent unions, small businesses and consumers, etc., were examined. Finally comes the conference directory of delegates, advisers and committees, and other relevant information. *J. N. Agate.*

OCCUPATIONAL DISEASES CONTROL

(Baltimore City Health Department, Industrial Health Series, No. 1.)

This is a symposium of the schedule of occupational diseases, the rules and regulations for their control and prevention, and the penalties for failure to comply with these regulations, which apply under the Occupational Disease Law of the State of Maryland, U.S.A. Sections on the reporting of occupational diseases and the industrial hygiene services available in Baltimore are also included. Under the Schedule of Occupational Diseases employees in certain listed processes, or subjected to a risk of certain specific diseases, are deemed to be employed in extra-hazardous work, and compensation is then payable for any disability or death caused thereby. The listed diseases are: anthrax; lead, zinc, mercury, phosphorous and arsenic poisoning and their sequelae; wood alcohol poisoning; poisoning by benzol or nitro-, hydro-, hydroxy- or amido- derivatives of benzene; by carbon bisulphide or any sulphide, by nitrous fumes, by nickel carbonyl, or the sequelae of any of these; poisoning by dope, formaldehyde and its preparations, hydrocyanic acid, chlorine, ammonia, cadmium or manganese; chrome ulceration or dermatitis or the sequelae; epitheliomatous ulceration from any of the usual industrial agents; glanders; compressed air illness or its sequelae; certain miners' diseases; cataract in glassworkers; poisoning from radium, radioactive substances or *x* rays; poisoning from methyl chloride or other halogenated hydrocarbons, carbon monoxide, or sulphuric, hydrochloric, or hydrofluoric acids; certain lesions due to petroleum products and their fumes; disability from blisters, abrasions, bursitis or synovitis; dermatitis venenata; silicosis; asbestosis. Recent additions to the list are: respiratory, gastro-intestinal or eye disorders due to contact with antigenic substances; occupational tularaemia, brucellosis, psittacosis, leptospirosis, rabies, or Rocky Mountain spotted fever, and selenium, tellurium, and fluorine poisoning. In each case the process or occupation in which the hazard can be expected to occur is laid down. *J. N. Agate.*

THE TOXICITY OF MOLYBDENUM

By L. T. Fairhall, R. C. Dunn, N. E. Sharpless, and E. A. Pritchard

(U.S. Public Health Service, Public Health Bulletin, No. 293. 1945.)

Molybdenum steels are used for munitions, high-pressure boiler plates, and tool-making, and are extremely tough; molybdenum can be used to make alloys with tungsten and chromium; in chemistry it is used as a catalyst, and in the making of pigment colours of great brilliance; it is used in vitreous enamelling, in high temperature furnaces, and in radio valves. The metal is hexavalent and occurs naturally as lead and iron molybdates, but mainly as the sulphide, molybdenite, which physically resembles graphite. There is no hazard to be expected in the mining of the ores, but in their smelting there is exposure to calcium molybdate dust, and in the rolling of hot billets exposure to molybdic oxide fumes.

Estimation of molybdenum is best done by a colorimetric method based on the red-orange colour given with thiocyanites in acid solution by reduced molybdenum, with an extraction medium to intensify the colour. Details of the method are given in this report. Molybdenum is widely distributed, although only in fractions of a milligramme per kilo, in both plant and animal tissues, including human liver and spleen and carcinoma tissue of the breast. Some plants contain enough to be injurious to herbivores. Previous animal experiments on the toxicology are reviewed; these had been confined to ingestion and intravenous injection.

In the experiments reported here, dosage was given by inhalation to simulate toxic hazards in industry, by ingestion to follow the course of absorption and excretion, and by intraperitoneal injection to determine if the substances are irritant or inert. Molybdenum trioxide, calcium molybdate, and ammonium molybdate proved fatal to rats and guinea pigs when given in doses of 1,200 to 6,000 mg. per kilo, if given orally into the back of the animal's throat by a syringe; with increasing dosage there was increasing storage in the liver and other tissues. Doses of one-tenth of the above produced fewer fatalities; 6,000 mg. of molybdenum as molybdenite did not cause death, and this was also the case in exposure to the dust of the molybdenite. Dust of this compound and of calcium molybdate and molybdenum trioxide were introduced into a chamber by means of an elutriator. The last two reagents in concentrations of 5 mg. per cubic foot were very irritating, caused loss of appetite, loss of hair, diarrhoea, inco-ordination, and death. Animals were exposed to molybdenum trioxide fumes produced by arcing electrodes of the metal, but no fatalities occurred, which was unexpected. Experiments on the solubility of molybdenum trioxide were then done to elucidate the problem; in the fume state it is most soluble.

Intraperitoneal injection of soluble molybdenum compounds, particularly molybdic oxide and ammonium molybdate, produced a high mortality in guinea-pigs when given in doses of 400 to 800 mg. per kilo. Respiratory paralysis and convulsions were seen. Radiographs suggested that molybdenite and calcium molybdate remained *in situ* but molybdenum trioxide was dissolved and redistributed. Storage of molybdenum compounds is transient and takes place mostly in the kidneys and bones, and to some extent in the spleen. Absorption

and excretion are both rapid after dosage by ingestion; the blood level rises after about 100 minutes. Examination of blood and urine will indicate the extent of the absorption of molybdenum.

Parallel experiments on the relative toxicity of chromates, tungstates and molybdates of sodium by intraperitoneal injection show that the molybdate is the least, and the tungstates the most, toxic. Histological studies throughout these experiments show minimal changes. The liver shows fatty infiltration and small focal necroses. The findings suggest that the compounds of molybdenum in general are of low toxicity. In industry the worker should be protected from inhaling large quantities of the more soluble compounds.

J. N. Agate.

A MEDICAL STUDY OF THE EFFECT OF TNT ON WORKERS IN A BOMB AND SHELL LOADING PLANT

R. F. Seivers, A. H. Lawton, F. Skoog, P. A. Neal and W. F. von Oettingen

and

REPORT OF FATAL CASE OF APLASTIC ANAEMIA

R. L. Stump, A. R. Monaco and R. F. Sievers

Publ. Hlth. Bull., Wash., No. 291, 1945. (Pp. 98; refs. 70).

This is a painstaking study of 250 men and 103 women in contact with TNT, and of 55 men and 50 women controls, but little new is added to existing knowledge. For those who are unfamiliar with the methods employed in filling bombs and shells, the description and photographs of the process form the most valuable part of the report. No cases of severe TNT poisoning were seen, but 21 workers were considered to be suffering from early or mild TNT intoxication. No single test is diagnostic of TNT poisoning in its early stages. Workers exposed to TNT, but not showing signs of intoxication, showed a decrease in the haemoglobin, haematocrit reading, and red blood count, slightly increased in the icterus index, and urinary excretion of coproporphyrin. Snyder and von Oettingen's (*U.S. Publ. Hlth. Bull.*, No. 285, 1943; and *J. Amer. med. Ass.*, 1943, **123**, 202) test for 2·6 di-nitro-4-aminotoluene (a metabolic product of TNT) in the urine, besides being quantitative, proved more sensitive than Webster's test as an index of absorption. The diet of all workers was good, but symptoms were more common in non-milk drinkers; the consumption of poly-vitamin capsules offered no protection. Men were more frequently affected than women, and the increased incidence of complaints in men was thought to be independent of the fact that the average duration of contact for men (8 months) was longer than that for women (3 months) or that men were employed on more dusty jobs. Loss of weight, if accompanied by other signs of TNT intoxication, was considered to be due to TNT. Compared with the control group, exposed workers complained more frequently of fatigue, malaise or drowsiness, anorexia and vomiting, abdominal pain, headache, dizziness, and polyuria. Cyanosis was observed among 68% of the exposed men and 36% of the exposed women, and pallor in 45% of the men and 41% of the women, but the pallor is unrelated to blood changes. Forty per cent. of the exposed groups complained of dermatitis at some time (usually in the first month). There were no significant cardiac changes.

A fatal case of aplastic anaemia following TNT hepatitis in a 43-year-old white male, after slight exposure to TNT, is reported. After he had complained of nausea, anorexia, constipation, sore throat, lumbar pains, burning of the eyes, and burning on micturition, it was discovered that he was jaundiced and that his liver was enlarged. The icterus index was 100 and Webster's test negative. The red cell count dropped from 4,010,000 to 2,730,000 per c.cm. of blood, and Hb from 15·0 to 7·5 gm. in 75 days. The white cell count fell to 2,400 per c.cm., and platelets disappeared. Folic acid was given too late to be effective. Histological section of the liver showed almost complete disappearance of the liver cells, but the bone marrow appeared of normal cellularity, with a few monoblasts and occasional erythroblasts. Granulocytes were rare and primitive.

T. A. Lloyd Davies.

AN EXPERIMENTAL STUDY OF RATIONING

By R. A. McCance and E. M. Widdowson (1946)

(Spec. Rep. Ser. Med. Res. Coun. Lond. No. 284. pp. 61. 1*s*.)

This is an account of an experiment carried out by eight people in which they rationed themselves to a diet which they considered might become necessary for the whole population if unrestricted submarine warfare was successful. Government departments are apt to attempt to suppress scientific information, and this activity can, in wartime, always masquerade under security. Now that this excuse can no longer be upheld, the Medical Research Council has adhered to its principles that scientific work must be published. Readers may judge for themselves how valid was the security excuse for suppressing this simple experiment which might have been repeated by anybody at any time, and give thought to the unhappy results which may occur when ministers and their bureaux have complete control of everything. The diet envisaged included 4 ozs. fat, 5 ozs. sugar including jam, 35 ozs. milk, 4 ozs. cheese, 16 ozs. meat, 6 ozs. bacon. The bread was made from flour of 92 per cent. extraction, while the consumption of potatoes and vegetables was unrestricted. During strenuous exercise, which included cycling 90 miles a day, or walking 38 miles a day in the Lake District, they sometimes ate as much as 3 lb. of potatoes, the calorie value of the diet being between 3,000 and 4,000 a day. Throughout the experiment all food was weighed and analysed in detail, and during three separate weeks faeces and urine were collected and analysed also in the greatest detail.

The experimenters kept to the diet for three months and at the end were very fit and could carry out satisfactorily, and without undue fatigue, most severe exercise tests, requiring a very high expenditure of energy. The results certainly show that young adults could have maintained a high standard of health under dietetic conditions much more austere than those actually experienced in the war. The authors could see no justification for the enormous amounts of meat in the rations of the soldier, and considered that troops, in this country, at any rate, would have been just as well served with bread made from lightly milled flour, more potatoes if necessary, and less meat. From these experiments it is not possible to say how children and old people would have fared under these dietary conditions, but it is probable that those who could eat the food allotted would have maintained good health.

K. M. A. Perry.

ABSTRACTS

Occupational Rheumatic Fever. CASTRO, R. E. (1945). *Rev. argent. Rheumatol.*, **10**, 260.

The writer describes an illness in a quarry worker aged 18. When he was seen on the third day he was febrile with sweating, pains in the head and back, and fleeting pains in the joints. He had chronically infected tonsils, which had been inflamed for a short time before. He had been given a sulphonamide drug, but treatment was changed to salicylates, whereupon his condition improved. The tonsils were removed after convalescence.

The writer suggests that, as the patient was chilled at his work, and chilling is known to conduce to respiratory infections and sore throats, the illness may have been occupational in origin. *G. C. Pether.*

Resuscitation of Severely Wounded Casualties. LALICH, J. J., and MASON, J. M. (1945). *Surgery*, **18**, 741.

This article was written after two years' experience in the treatment of casualties in North Africa and Italy. Severely wounded men were classified as 'non-transportable.' These were chiefly patients with penetrating wounds of the abdomen, pleura, and spine, traumatic amputations, and severe compound fractures or vascular injuries. Non-transportable cases were further grouped under the following heads: (1) those in whom trauma and haemorrhage were the main cause of a failing circulation; (2) those who suffered also from cardio-respiratory embarrassment; (3) those in whom infection was already present. On admission the state of the circulation was assessed by reading the blood pressure. Three grades were recognized: (1) those in whom the blood pressure was 90 mm. Hg. or over, but in whom circulatory failure seemed imminent; (2) those in whom the blood pressure was under 90 mm. Hg.; and (3) those in whom no ausculatory blood pressure could be detected. It was generally found, especially in the last group, that the immediate prognosis depended on the injured man's response to intravenous therapy. If the blood pressure rose to about 70 mm. Hg. or more, immediate danger was generally over. Although the authors put great trust in the blood pressure reading as a guide to response to therapy, they stress the fact that in many cases a fall occurs during or after operation, and that, if more blood is not given when this second fall occurs, the result may be fatal. There is a table showing the response of this secondary fall to intravenous therapy. After discussing the clinical manifestations of shock, the authors describe their routine management of shocked men. Heat was not used, but the men were stripped and placed between dry blankets. Oxygen was used, but only if indicated. Further treatment depended on the blood pressure. If this was over 90 mm. Hg., 1,000 c.cm. of blood were given slowly over a period of two hours. When the blood pressure was under 90 mm. Hg., blood was given more quickly—the first 500 c.cm. being administered in 30 minutes. Those with no blood pressure were given 2,000 c.cm. of blood and plasma, the rate being 500 c.cm. in 10 minutes until the pressure rose to 70 mm. Hg., when the rate was diminished to 500 c.cm. in half an hour. The authors think that the biggest factor in the production of shock in wounded men is loss of blood, and that the best treatment is to replace that loss as soon as possible.

E. C. B. Butler.

Lead Excretion in the Bile after Intravenous Injection. BLAXTER, K. L., and COWIE, A. T. (1946). *Nature*, **157**, 588.

The authors describe experiments with sheep. Soluble lead salts were injected intravenously, and only from 5–8 per cent. were excreted in the five days following injection. Excretion in the urine was immediate, and corresponded with a high plasma level, but faecal excretion was delayed four or five days. Lead acetate in saline—150 mg.—was slowly injected into one animal which had had complete biliary drainage from a modified Schoregge operation. Excretion in bile, faeces, and urine was measured. Both biliary and urinary excretion were enormously increased, with a close relationship between them and the blood figures. The sheep died of uraemia on the seventh day, and autopsy showed marked renal damage, of which there had been evidence from the fourth day. Blood examinations showed anaemia and nitrogen retention. Over the six days, 7·4 per cent. of the lead was excreted; and of this, 17·1 per cent. was in the urine, 80·7 per cent. in the bile, and 2·2 per cent. in the faeces. The maximal percentage of biliary lead excretion was on the fourth day, and the delay may possibly have been related to liver cell breakdown. Anuria of 24 hours on the fourth day possibly accounted for the high concentration then. The view that the bile is the chief channel for lead excretion in the intact animal is supported by these experiments—which is interesting in connexion with Anderson's observation on the lead content of biliary calculi.

G. C. Pether.

Symposium on Burns from the Hartford Circus Disaster. 1. Management of civilian disaster burns. WELLS, D. B. (1946). *Occupational Med.*, **1**, 99.

The author reviews in detail the lessons which he and his colleagues learned in dealing with a large number of casualties. The most important points can be summarized as follows. (1) That the treatment of burns is a very complicated problem, and should be undertaken whenever possible by a team of men who are experienced in the subject. (This is already well recognized in this country.) (2) That first-aid treatment should be minimal; the wounds should be covered over by a clean dry cloth; no ointment should be applied. (3) That local authorities should always know the capacity of all the hospitals in their district so that in the event of a major disaster no one hospital would be overcrowded with cases. (4) That each hospital should, whenever possible, keep aside a ward with a number of empty beds—from 20 upwards—equipped with the necessary apparatus for treating shock. Sufficient stores of plasma and blood should also be available at short notice.

All cases were at once put under the care of the burns committee of the hospital. They were nursed together and all received the same treatment. Eighty-six patients came to the hospital. Of these, 67 were admitted, of whom 54 were burned; 3 of these died. Because of previous plans there was no confusion at the time of the accident, and plentiful supplies of plasma and blood were in store. Preliminary treatment consisted in intravenous morphia, supplemented when required by intranasal oxygen. Shock was further treated by plasma, controlled by repeated haemoconcentration estimations. The burns were not cleaned or excised; each was covered by strips of sterilized petrolatum gauze overlaid with gauze and firm bandages. The limbs were immobilized by newspaper splints. Later, local treatment consisted in the application of a bland ointment with massive protective dressings. Delayed skin grafting was done where required. No local chemotherapy was employed in any case. The author believes that the technique of dressing is more important than what is actually applied. His criteria of a successful dressing are good protection from contamination, prevention of capillary leakage, immobilization, and infrequent dressings. The results were said to be good, but no figures are given to indicate

the rate of healing or the extent of infection. This paper is interesting because it shows the swing of the pendulum from coagulation therapy back to the application of simple ointments. The control of sepsis is left entirely to systemic chemotherapy.

2. Esprit de Corps among Catastrophe Victims. ROOT, M. T.

All the patients were under the care of the Burns Committee, and there was no differentiation in social status. As far as possible all victims were nursed in the same wards. The result appeared to be a very high morale which the author attributes to three main factors. (1) Common injuries and uniform treatment. Sufferers from a common injury assisted one another to bear the pain; this was especially true if dressings were being done. (2) Individual security. There were plenty of nurses for each patient; and the full co-operation of the social service department ensured that all home worries could be cared for. (3) Patients felt they were getting the very best treatment. The burn committee, which often included many distinguished visiting physicians and surgeons from all over the country, made daily rounds. As a result of the sense of security there were hardly any psychiatric problems.

This short paper illustrates the advantages which occur when patients suffering from similar accidents can be treated together; the value of a good social service attached to each hospital is brought out.

3. The Laboratory in the Burn Catastrophe. KENDALL, R. E.

The immediate problem was the estimation of haemoconcentration. The determination of this by centrifugation (haemotocrit) was impracticable, because venipunctures were often impossible owing to the extent of the burns or the collapsed state of the veins. Blood was obtained from the ear or finger, and the copper sulphate method of Phillips and Van Slyke was used. This method proved rapid and accurate. The chief observation noted was that haemoconcentration tended to persist for 48 hours in most cases; this proved a more useful guide to the state of the patient than either the blood pressure or the pulse.

4. Bacteriostatics. LEONARD, J. C.

Soon after admission all patients were put on sulphadiazine by mouth. The dose varied with the age of the patient. The weather at the time was humid; this, combined with the haemoconcentration already present, proved dangerous. Many patients showed red blood cells, and a few rapidly developed haematuria. All sulphonamides were stopped after 48 hours. In the author's opinion, routine administration of sulphonamides in cases of severe burns is dangerous. Penicillin was tried. In adults the dose varied from 5,000–35,000 units three-hourly, while children received from 5,000–20,000 units. The number of days this treatment was continued is not stated. Cultures taken from the wounds showed moderate to heavy growth of gram-negative bacilli, staphylococcus aureus, and non-haemolytic streptococci. One fatal case developed severe infection with anaerobic streptococci which were resistant to penicillin and the sulphonamides. No penicillin was used locally.

It is evident that considerable local infection occurred in the burns; one might think that local chemotherapy would have lessened its occurrence. Anaemia was general, and was treated by whole-blood transfusions, and by iron orally.

Treatment of hypoproteinaemia consisted in giving a high protein diet to all patients. This was reinforced by soya bean and amigen. In certain very ill patients, feeding was successfully performed by means of a duodenal tube.

This article shows that systemic chemotherapy alone will not control the local infection of burned tissues, especially when the dead tissues have not been excised.

E. C. B. Butler.

Diagnosis and Primary Treatment of Injuries of the Hand. BROWNE, W. E. (1945). *R. I. med. J.*, **28**, 875.

The author reviews the incidence of hand injuries in industry. He claims that 70 out of every 100 industrial accidents affect the hand. Despite many precautions, it is probable that the circular saw is still the worst offender. Diagnosis of hand injuries is discussed, and there is a short account of the more important anatomical features of the hand. The anatomy of the thumb is described in detail in order to facilitate the diagnosis and repair of injuries to it. In a discussion on the *x*-ray diagnosis of bone injuries, it is emphasized that *x* rays should be used not only when the injury first occurs, but also later on, if there is sepsis, so that osteomyelitis may be found or excluded. The anatomy of the median and ulnar nerves is described, together with the common sites of injury about the wrist. It is stressed that nerve injuries should be diagnosed by motor rather than by sensory signs, as the latter are often misleading. The blood vessels of the hand are enumerated. The author says that all bleeding points should be controlled before the skin is sutured, since haematomata are liable to produce skin sloughing or infection; that all wounds of the hand should receive treatment as soon as possible in order to avoid the serious complications arising from infection; and that the best method of cleansing an injured hand is by irrigation with 100 quarts of warm normal sterile saline.

Exact diagnosis is considered essential before the patient is anaesthetized for operative treatment. All wounds must be excised according to the usual principles; foreign bodies should be removed. The author says that avulsed skin can sometimes be resutured. He has found that secondary skin grafting is preferable in cases of pulp avulsion, but that all exposed tendons should be immediately covered by skin flaps or grafts. He finds that, if the wound is clean, primary nerve and tendon suture is often possible; in other cases the proximal end of the tendon or nerve should be anchored to the skin by means of a silk suture. This important step prevents retraction of the divided end and makes secondary suture much easier. No through-and-through drainage should ever be employed, as the drains may press on tendons and cause necrosis.

Immobilization is generally needed. The hand should be placed in the functional position in some form of a 'cock-up' splint. The author does not use local chemotherapy, but relies on systemic treatment in cases of threatened or obvious infection. This paper is full of common sense. Operative details are omitted, and emphasis is rightly laid on the principles of early diagnosis.

E. C. B. Butler.

Toxic Effects of 2,2–bis (p–Chlorphenyl) 1,1,1–Trichlorethane (DDT) in Man. CASE, R. A. M. (1945). *Brit. med. J.*, **2**, 842.

With care there is little hazard for humans in the use of preparations of DDT. It may be dangerous in oily solutions and when dissolved in acetone, and it is now being incorporated in paints and distempers. DDT is toxic to a variety of mammals, causing liver damage, leucocytosis and changes in the central nervous system. A case of human intoxication from DDT in solution of acetone and in powder form had previously been reported, showing subjective symptoms, prostration and muscular twitchings. An investigation is described in which the toxicity of a DDT distemper was studied because it was to be used in hot, cramped, Service conditions where contact with oil and unclothed skin might be considerable. Two volunteers were placed in a steel chamber for 48 hours at a time; they wore shorts only, and the atmospheric conditions could be varied at will. The chamber was first coated with ordinary distemper, then with the same distemper with 2 per cent. DDT., and subsequently with lubricating oil. Toxic effects were produced, showing as increased red-cell destructions, and increase in reticulocytes, a lowered haemodynamic index, neutropaenia with immature white cells appearing in the

films, and indicanuria. Symptoms were lassitude, heaviness, aching of limbs, unilateral deafness, pains in the joints, yellow vision, and an irritable and apprehensive mental state. There were slight neurological signs, but electro-encephalograms were normal. There was a transient fall of blood pressure. Both subjects were normal again within 33 days.

Full haematological investigations, with leucocyte counts, and tests for indicanuria, might be used in the early diagnosis of DDT poisoning. There is a definite hazard for man, but the special conditions of this experiment are emphasized. *J. N. Agate.*

Acute Yellow Necrosis of the Liver following Trilene Anaesthesia. HERDMAN, K. N. (1945). *Brit. med. J.*, **2**, 689.

This is a report of a case thought to be the first of its kind. A man aged 28 undergoing tarsorrhaphy after cordite burns of the face was given pentothal followed by gas, oxygen, and trilene. On the fifth day there was vomiting and hepatomegaly, on the sixth there was jaundice, and on the twelfth day, when the liver had become smaller, the patient died. The total quantity of trilene used was 2 fl. oz. over a period of 4½ hours of open circuit; 0·8 gr. of pentothal having been used after the usual premedication. The same trilene had been used for other patients without ill effect. Autopsy showed acute yellow necrosis, a liver two-thirds the normal size, subendothelial haemorrhages, and ascites. Possible reasons for the occurrence are discussed, and it is suggested that patients due for prolonged trilene anaesthesia should have intensive glucose administration. *J. N. Agate.*

A Fatal Case of DDT Poisoning in a Child. HILL, K. R., and ROBINSON, G. (1945). *Brit. med. J.*, **2**, 845.

A child of one year and seven months accidentally drank 1 oz. of 5 per cent. DDT in kerosene. Coughing and vomiting took place in 10 minutes, and within 90 minutes the child was comatose and in convulsions; within 4 hours he was dead. At autopsy there was oedema and congestion of the brain, oedema of the lungs, and an enlarged spleen. Two experiments were done on baboons to determine whether the kerosene alone, or the DDT in kerosene, was responsible. In both experiments the monkeys, when given the latter, became ill, and one of them died of pulmonary oedema. Kerosene alone had no such effects. Two dogs with skin lesions were sprayed with 5 per cent. DDT in kerosene; both died of exhaustion in two months. The lethal dose for a child can be calculated at about 150 mg. per Kg. of body weight, and it appears that, if DDT is given in organic solvents by mouth, the human is more sensitive than the experimental animals. The symptoms and signs of acute poisoning in the child and the monkeys agreed with those noted by other observers in their animal experiments. The dogs succumbed to chronic intoxication with liver and kidney damage. *J. N. Agate.*

'Acroparasthesia' and so-called 'Neuritis' of the Hands and Arms in Women. WALSHE, F. M. R. (1945). *Brit. med. J.*, **2**, 596.

An unusual number of women complaining of numbness and tingling of the hands has been seen. The women fall into two groups: the middle-aged, previously unaccustomed to heavy household duties, and the younger ones rearing children or doing part-time factory work as well. The onset is gradual, and the symptoms are worst in the morning on waking, persist to a less degree by day, and increase towards evening when the woman is fatigued. Burning pain may wake her at night. The condition fluctuates month by month. Response to treatment is uniformly disappointing, but some patients have said that a holiday improves them. They are usually chronically fatigued, and—though this is the general tendency in all middle-aged women—have an atonic musculature and a shoulder girdle set abnormally low. Neurological signs are usually negative, apart from tenderness of extensor muscles and those of the thumb. Radiological abnormalities are seldom found. There is probably no connexion with multiple symmetrical peripheral neuritis ('polyneuritis'). Constant aetiological factors are fatigue, debility and heavy use of the upper limbs; and the suggestion here is that the condition is a rib-pressure syndrome due to an altered anatomical relationship between a normal thoracic outlet and upper-limb girdle, pressure and traction being exerted on the lower trunk of the brachial plexus. Most other rib-pressure syndromes, due to abnormal first or cervical ribs, are unilateral and asymmetrical. Treatment consists of rest, and rest alone, however impracticable the suggestion. Patients should have a week in bed, cease heavy work, and have the arms in slings wherever possible. *J. N. Agate.*

Sulphaemoglobinaemia due to Aniline Deviations. LAZARUS, S. (1945). *Brit. med. J.*, **2**, 565.

Sulphonamides have frequently been reported as causing sulphaemoglobinaemia, but less notice has been taken of simple antipyretics. Acetanilide is relatively toxic and is scheduled in the Poisons List (1937), Part 1. Phenacetin is less toxic, but is obtainable without restriction. Two cases of sulphaemoglobinaemia are reported, one in a housewife accustomed for years to taking from 8·4–63 gr. of phenacetin daily (in a proprietary tablet) for intractable psychogenic headaches. She had a slate-grey cyanosis; her blood picture was normal, but sulphaemoglobin was discovered spectroscopically. The second case was of a man who had taken 240 gr. of phenacetin and 120 gr. of acetanilide over two weeks; the findings were the same. Both recovered successfully, but the cyanosis, unlike that of methaemoglobinaemia, was slow to disappear. Patients may deny that they have given themselves such drugs as these. Aniline derivatives are probably oxidized to a phenyl-hydroxyl amines and para-aminophenols which affect haemoglobin in the presence of traces of sulphide. It is possible that coloured oxidation products of para-aminophenol are circulating and contribute to the cyanosis. *J. N. Agate.*

Medical Care in the Coal Mining and Steel Industries of Nova Scotia. LYNCH, J. G. B. (1945). *Canad. med. Ass. J.*, **53**, 542.

Anticipating the establishment of health insurance in Canada the author advocates that a council be set up to investigate medical practice in the coal mining and steel industries in Nova Scotia. After describing the present service available in both industries and the steps which have been taken to bring them to their present standard, he contrasts the contract practice in the coal mining industry with the altogether different organization in the steel industry. In the former, the doctor's life is often too strenuous to allow him to give complete, careful, scientific medical attention to those under his care; the dispensing of drugs is universally disliked; and the annual cost of medical service to the miners is approximately $266,585, plus hospital costs of $300,000. In the steel industries, on the other hand, the employer covers the total cost of medical aid, and industrial accidents are cared for by a medical and nursing staff, who have at their disposal up-to-date hospitals, dressing-stations and ambulance service. In contrast, the colliery districts still rely on first-aid dressing stations under the charge of first-aiders whose efficiency is sometimes questioned. The fact that the compensatable accidents to workmen per 10,000 man-days worked in 1944 in all collieries was 7·7, compared with 0·45 in the steel industry, emphasizes the good results of the type of practice in the latter industry. The author says that miners want a 'clinic' type of practice, but he hopes that medicine will not be allowed to become a 'political football.' *A. Lloyd Potter.*

Experimental approach to the Nervous Symptoms of Manganese Poisoning. VAN BOGAERT, L., and DALLEMAGNE, M. J. (1945). *Mschr. Psychiat. Neurol.*, **111**, 60.

Few autopsies after manganese poisoning are recorded, and experimental work is scanty. The symptoms of poisoning usually come on quickly and need not be described in detail. Some writers have used the term 'Manganese Parkinsonism.' The authors discuss some previous work, and conclude that, though manganese tends to disturb the strio-hypothalamic functions, and sometimes other tissues, intoxication extends beyond the visible changes. After referring to Mella's work with monkeys—in which Mella produced a choreiform, and later a Parkinsonian syndrome, thought to be due to changes in the corpus striatum—the writers describe their own experiments. Mella gave his animals manganese chloride intraperitoneally. The authors poisoned one of their monkeys (*Macacus rhesus*) by using an aerosol according to Dautrebande's method. Another animal, used as a control, was given manganese in its food. The period of exposure to the aerosol was one hour daily. The first signs of poisoning were sudden, sharp movements, nervousness, and periods of torpor alternating with movement and frequent yawning—the latter apparently associated with increasing anoxaemia. Later there was alternate extension and flexion of the upper limbs, and spreading—with rapid trembling—of the fingers and toes. Still later trembling became almost continuous and took place at intervals after exposure to the poison had ceased. Sometimes the chest was thrust out and the head thrown back. When lying down the animal would shake all over as if from cold. After a hundred days' exposure the animal was rested, and three weeks later the nervous signs described had ceased. Cyanosis, which had been observed previously, had gone; and yawning was less frequent.

Five months passed, and then somewhat different nervous symptoms became evident. Any voluntary movement seemed to set up fresh manifestations. Tremors, though interrupted, again developed. The head was held to one side and the animal walked with a broad spread. There was much uncertainty of hand movements, and apparent loss of postural sense. When the monkey sat down the gross tremors of the limbs stopped. After two weeks the animal could barely walk or sit up without support from the hands. Every movement was followed by such numerous and violent involuntary movements that it could not rest at all. When feeding, the animal lay on its belly. It looked tired and indifferent. A month later tremors became less frequent, occurring only when the animal changed position. Gait was uncertain, with some stiffness of the legs. One month after this, paresis of the legs was noted, and in the next eight weeks the monkey moved less and less.

On histological examination, degenerative changes were seen in the chiasma. In the spine there were changes in the anterior columns in the lumbar region, and in the anterior cornua; similar changes were seen in the dorsal and cervical cord. There were older sclerotic changes in the columns of Goll and Burdach at the fourth lumbar vertebra, and in Goll's column alone from the second dorsal to the second cervical. A Spielmeyer section through the fifth lumbar vertebra confirmed the demyelinization of the entire posterior column. A section at the second lumbar vertebra showed similar changes in Goll's column. Degenerative changes were seen in the olivary body, and certain minor changes in other parts of the bulb close to the surface. The cerebellar cortex was much damaged, involving both the Purkinje and granular layers. There were slight changes in the white matter and in the dentate nucleus. The internal organs exhibited no notable changes.

The control animal was given 8·2 g. of manganese sulphate in its food over a period of six months. This animal never showed any sign of involvement of the central nervous system. It died quietly six months after manganese administration was stopped. Even with the sensitive method of Kahane, no manganese was found in the heart, liver, lung, brain, or bones; this was presumably because of the long period which elapsed after poisoning had ceased. There were no pathological changes other than a degeneration of the myocardium which was difficult to interpret; no conclusive sign of manganese poisoning was seen.

Reviewing other work, the authors decide that hepatic and renal changes are directly related to elimination of the metal, which is largely excreted in the bile. The writers do not think that the changes in the nervous system are essentially secondary to those in the liver, and doubt if the conclusions of Charles have any clinical or experimental foundation. They suggest that the combination of a cerebellar-dentate atrophy with a mild anterior poliomyelitis is reminiscent of a similar combination in man, described by Welte. It also resembles the combination of hereditary ataxy and progressive amyotrophic spinal disease described by Lelong and others. *G. C. Pether.*

The Psychiatric Salvaging of Industrial Manpower in Wartime. SELIGER, R. (1945). *Southern med. J.* **38**, 799.

At the John Hopkins Psychiatric Clinic in 1943, 71 warworkers were treated, and 56 of these returned for follow-up. More than 50 per cent. were salvaged through psychiatry, and were productively employed. Experience showed that many patients, given some anchorage, support, and common-sense therapy, can be astonishingly well in spite of organic, psychotic, genogenic (i.e. constitutional disorder such as dullness or effeminacy), or neurotic disabilities. Causes of breakdown were found in both the home and the industrial environments, and were usually clear cut. Either these could be alleviated, or the individual could be taught to be productive in spite of them. In some cases special techniques, such as rapid analysis based on Rorschach testing, were useful; so also were letters to employers and interviews with the spouse. It was not necessary to review infantile traumata and the like. Direct contact with the employer was useful in getting the patient back to work in the same or in a different capacity, and also of ridding him of fears that the employer would think him crazy or malingering. Where changes in the industrial situation led to inevitable changes of job, the reasons were explained: otherwise the worker was apt to feel inadequate and 'pushed around.' Trained psychiatric workers were interchangeable, and patients got well without depending on direct personal rapport with any one individual.

These results suggest possibilities for further, deeper investigations by clinics and personnel departments regarding the causes and modifiable elements of some behaviour and emotional problem-illnesses in industrial workers, especially in a society which depends on the productive vitality of its members. *E. A. Larkin.*

New Data on the Use of Aluminium and of Alumina in the Prophylaxis and Treatment of Silicosis. POLICARD, A. (1945). *Presse méd.*, **53**, 605.

The author reviews early work in this field and makes reference to the observations of Jötten, who, in 1933, noted that the sandstone particles derived from stone found in the Eifel were relatively harmless when compared with those of the Palatinate. This was attributed to the fact that the former had a coating of alumina round the silicate core, whereas the latter had none. He then describes the work of the Canadians, Denny, Robson, and Irwin, who showed that the addition of 1 per cent. of metallic aluminium to a silicate dust prevented the development of pulmonary lesions by the latter. They thought that the aluminium particles were oxidized in the tissues affected by the dust, whereupon a film of aluminium hydrate covered the silicate particles and made them harmless.

The author, working with J. Rollet in 1939, was able to demonstrate a similar reaction when the two substances were injected into the cornea. A contradictory note was struck by Jötten in 1940. He found that aluminium

dust did not prevent the development of silicotic sclerosis, but that aluminium silicate could do so. Belt and King in 1943 also obtained negative results, but their methods were doubtful.

In 1944 Gardner, Dworski, and Delahant had similar results, using aluminium hydrate instead of metallic aluminium. By mixing this with silicate dusts before inhalation it was possible to prevent the development of lesions in the lungs.

The hydrated oxides of aluminium are of many different kinds and they are not fully understood. L. U. Gardner found that some had a marked protective power, whereas others were relatively useless. It seems, then, that more constant results can be obtained with aluminium metal.

But it is important to discover whether this is itself quite harmless. In 1940 the writer made experiments which seemed to confirm the view that it was innocuous. Inhalations of as much as 20,000 particles per cubic centimetre prolonged for some weeks did not cause any pulmonary lesions in rats.

Investigations have also been made in workmen exposed to aluminium dust or the dust of aluminium bronze. There is some doubt, but apparently these may cause bronchial irritation but not changes of silicotic type. This being so, it is not likely that the quantity of aluminium dust used as an antidote can do harm.

L. U. Gardner showed in experiments on guinea-pigs that small quantities of alumina were harmless when inhaled by normal or tuberculous animals, provided that the quantity was not excessive nor inhalation too prolonged. But if too much were inhaled then the tuberculous lesions spread, though there was no destruction of tissue. Cicatrization of the lesions did not appear to be retarded. These changes were possibly to be explained by a blocking of the reticulo-endothelial system. In any case it is wise, before using aluminium, to exclude tubercle by radiographs.

Aurine, an aniline colour, if in contact with aluminium hydrate at pH 5·2 takes on a reddish tint. By using this dye one may demonstrate the adsorption of the aluminium on the quartz particles. But in some tests designed to show this, negative results have been obtained. Nevertheless the essential fact of the formation of a protective film is not in dispute. *G. C. Pether.*

Casualties Following Exposure to Zinc Chloride Smoke. EVANS, E. H. (1945). *Lancet*, **2**, 368.

Zinc chloride smoke in the open, as used for smoke screens, causes only minor symptoms, but the ignition of smoke generators in a confined space caused severe toxic manifestations; 70 people were affected, and there were 10 deaths. The exposure in the region of the generators was to ½ lb. per cubic yard. Seventeen cases were studied in detail; all complained of dyspnoea, pain or constriction in the chest and epigastrium, stridor, and lacrimation. Many had cough and blood-stained sputum, and showed a grey, earthy cyanosis. Subsequently an acute inflammation of the whole respiratory tract developed, leading to bronchopneumonia. In spite of severe symptoms, the auscultatory signs were throughout minimal. Recovery, when it took place, was slow, and occurred after the 7th to 10th day; it might be interrupted by a return of pyrexia or, as in one case, a lung abscess. Autopsies on two cases showed an inflammed, necrotic respiratory mucosa, subpleural haemorrhages, bronchopneumonia, and congested abdominal viscera. Deaths in the acute stage appeared to be from water-logging of the lungs. Treatment was by bed-rest, oxygen by B.L.B. mask—which failed to relieve the cyanosis—steam tents for the stridor, and, in spite of the acute respiratory distress, morphine with atropine for the general agitation. Milk feeds were given, and also sulphapyridine, 6 g. daily from the second day. The local effects are probably due to the astringent and hygroscopic action of zinc chloride, and pulmonary oedema was less evident here than in cases due to lung-irritant war gases; the respiratory tract was, however, more damaged. Respirators give complete protection. *J. N. Agate.*

Hospital, Industry and the Injured Man. GIRDLESTONE, G. R. (1945). *Lancet*, **2**, 765.

In this address the author first points out the essential weakness of the Workman's Compensation Act, that it causes the men sometimes to put their compensation before their desire to return to work. In such cases much time has been lost and many men have suffered moral deterioration. It is next claimed that modern methods of rehabilitation have largely overcome these disadvantages by attacking the problem as follows.

The Orthopaedic Hospital.—All members of the staff should be animated by a common purpose, which is to do their work efficiently and to see that everything possible is done for each individual patient. Ideally, a complete team of experts should be provided; but it is stressed that the most important factor in all hospital treatment is for the injured man to do as much as possible for himself. This is described as 'the mainspring of recovery.'

Works Management.—The management should co-operate closely with the hospital and facilitate the injured man's return to work by grading the amount that he is allowed to do.

The Man.—In the majority of cases the injured man will respond well to good hospital treatment, especially if it is combined with the personal interest of his firm. This has been found true both here and in the U.S.A. There still remains, however, the occasional delinquent who takes advantage of what is given him in order to prolong his incapacity. *E. C. B. Butler.*

Lead Poisoning and Haematoporphyrinuria. HUG, R. (1946). *Schweiz. med. Wschr.*, **75**, 322.

Haematoporphyrinuria may develop as the result of the toxic action of certain substances. It is identical with the inborn, familial condition, though this may be latent. It is suggested that the 'toxic' form occurs in persons with some predisposition to it. Sometimes infections provide the necessary trigger action. Among the substances which may cause haematoporphyrinuria, the sedative drugs—sulphonal and its derivatives, barbiturates and pyramidon—are very important. A case of lead poisoning in which this condition developed has been observed. The patient worked with lead paints in a porcelain works and used to lick his brush. The first symptoms were abdominal colic, vomiting, and constipation. There was also a rose tint in the urine and a positive test for urobilinogen. Lead poisoning was suspected. Very soon there was marked haematoporphyrinuria and other symptoms of lead poisoning, such as paraesthesiae, confusion, and lassitude. Paresis developed in the arms and legs. The patient became incontinent, and later died. While in hospital the patient passed little urine, and tests for urobilin and urobilinogen were consistently positive. Lead was found in the blood in quantities above the normal, though the amount in the stool was not above the normal. There was a slight secondary anaemia with punctate basophilia. In one week the urine contained from 2·6 to 8·7 mg. of coproporphryin, and from 2·6 to 15·3 mg. of uroporphyrin. The stool in one test contained 17·8 mg. per cent. of porphyrin. The mother's urine did not contain the pigment, and the urine of a cousin contained 0·1 mg. per litre. At necropsy there was oedema of the lungs with patches of pneumonia and smoke-grey colouration of the liver, but in sections only fatty change in the Kupfer cells. In the sciatic nerve the axis cylinders had disappeared in places. The lead content of liver and kidneys was normal, but the quantity in the marrow exceeded the normal figure.

There is much clinical similarity between lead poisoning and haematoporphyrinuria, and, when the intake of lead increases, the quantity of porphyrin in the urine also rises. In a normal urine there is 10–80 μg. porphyrin in the 24-hours' specimen, whereas in those exposed to lead, but who do not show any symptoms of poisoning, 15–30 μg. may occur. This early rise may be taken as an indication

of injury by lead. In recognized cases of lead poisoning there is from 500 to 8,000μg. Some writers suggest that the symptoms of lead poisoning are due to porphyrin formation and that this substance causes vascular spasm. There is considerable evidence against this view. The nervous symptoms are very dissimilar. In lead poisoning, apart from the encephalopathy, the prognosis is good, but in haematoporphyrinuria the outlook is grave.

The substances excreted are also unlike in the two conditions. In haematoporphyrinuria there is an excretion of uroporphyrin of the isomer series I or III. Both may occur together. In lead poisoning coproporphyrin III is found and uroporphyrin is rare and scanty. Vannotti showed that in all probability iron does not enter into the constitution of the porphyrin compound in lead poisoning, so that the porphyrin increase would seem to be related to the disturbed blood picture.

In acute cases of haematoporphyrinuria there is porphybilinogen or chromogen, but this is not found in lead poisoning. Lead poisoning and haematoporphyrinuria are thus two distinct conditions. It is not certain whether the porphyrins formed are themselves responsible for the symptoms of haematoporphyrinuria. Porphyrins have been shown, in experiments, to cause contractions of the isolated gut, but there is evidence to show that lead acts in a different way. In conclusion it is suggested that lead does not, as does sulphonal, release a latent porphyrinuria by some specific action independent of an existing diathesis. *G. C. Pether.*

Poisoning Due to Mobilization of Lead from the Skeleton by Leukaemic Hyperplasia of Bone Marrow. BROWN, A., and TOMPSETT, S. L. (1945). *Brit. med. J.*, **2**, 764.

Recently absorbed lead can be readily mobilized from bony trabeculae. The case is described of a compositor who presented with neurological signs, headache, intractable pains, thirst, and vomiting. The blood lead level rose from 280 to 455μg. per 100 ml. in two weeks, but the leukaemic blood picture did not develop until two months later. The leukaemia was of a subacute type, confirmed later at autopsy. It is noted that the symptoms of plumbism got worse, even though the patient was removed from lead exposure and given a diet conducive to storage. Though the blood changes followed the evidence of plumbism, the latter probably resulted from the marrow hyperplasia, which in leukaemia often precedes an abnormal blood picture, with consequent mobilization of lead. This may have been the reason for the low figures for lead in soft tissues at autopsy. A subsequent investigation has suggested that rarefying diseases of the skeleton may mobilize lead even after only slight exposure to the metal. *J. N. Agate.*

Occupational Eye Disease. MINTON, J. (1946). *Brit. med. J.*, **1**, 211.

Accurate definition of occupation has a bearing on ophthalmic diagnosis. A grinder may have a superficial corneal foreign body, but if the work is hammering or chipping the body may well be intraocular. Retinitis, palsies and retrobulbar neuritis may be due to industrial solvents. Many cases of superficial punctate keratitis occurred from H_2S exposure in the artificial silk industry and in the sugar industry. Keratitis also occurs from benzene and other varnish, and from shellac solvents; also from irritant vegetable substances which affect agricultural workers. Chronic symptomless corneal erosions are found in sandblasters, stonecutters, grinders, and lathe-workers. Bakers are subject to chronic conjunctivitis and blepharitis. Legge reported that 20 per cent. of glass workers had lenticular opacities from exposure to infra-red rays over 10–20 years. Protective goggles were invented, but furnace workers are still inadequately protected. Methyl alcohol causes optic atrophy, neuritis, or palsies; carbon bisulphide sets up optic neuritis, and so does arsenic; lead may cause neuritis or palsies. Carbon tetrachloride causes peripheral field amblyopia. Benzene and dinitrobenzene causes optic neuritis, and trichlorethylene both neuritis and retinitis.

Welders, through negligence over lowering their protective shields, suffer from 'arc eye.' The complaint can arise after only 6 hours' exposure; conjunctivitis appears first, and may go on to superficial punctate keratitis. Treatment consists of cold applications and guttae adrenaline 1: 2,000 with 1 per cent. helocaine. Some welders have chronic superficial keratitis with multiple erosions. Welding should be done in booths to prevent injury to adjacent workers, and welders' screens should be inspected regularly. Chronic conjunctivitis and keratitis result often from dusty workshops.

A new department of Industrial Ophthalmology has been set up at the Royal Eye Hospital, London, where the opinions of an engineer and safety officer are readily available. Corneal foreign bodies are the most numerous cases. Protective goggles are advised, and the various objections to them which workers raise are considered. Employers are co-operative. It is believed that more departments of industrial ophthalmology should be set up. *J. N. Agate.*

Observations on the Development of Silicosis. DAVIS, H. E. (1945). *Illinois med. J.*, **87**, 80.

All changes due to silicosis are visible radiologically, but minimal amounts of silica may be present in the lungs without causing changes. In order to produce changes the dust must reach the alveoli and, through a physical-chemical reaction, produce abnormal fibrous tissue. Only particles of the size order of 0·5 to 2·5μ are likely to produce changes. The protective mechanism of the bronchi excludes larger particles. Aluminium oxide may retard, and alkaline soap powders may accelerate, the usual reaction to free silica. Individual factors which affect the development of silicosis include frequency and depth of breathing, efficiency of the upper respiratory tract in eliminating dust, and the physiology and structure of the respiratory tract in a given individual. Environmental factors include the composition of the air-borne dust, the composition of the dust retained in the deeper portions of the lung and the amount retained, the particle size of the alveolar dust, the flocculating properties of the dust, the rate of phagocytosis, and presence of other materials. Inflammatory changes, such as tubercle, allergy, chronic bronchitis, and those changes due to inhalation of tobacco smoke and cardiac stosis, retard or limit ciliary activity and efficiency. Disability and the extent of the *x*-ray changes are not closely related.
K. M. A. Perry.

Relation of Fluorine to Dental Caries. ROBINS, C. J. (1945). *Connect. State med. J.*, **9**, 855.

The most common clinical manifestation of dental fluorosis is the appearance of a brown stain and, occasionally, grey or chalky-white spots of varying sizes and intensity, as an integral part of the tooth structure. The defect is found chiefly on the upper six anterior teeth, and is due to a faulty formation of enamel. The earliest investigation in Colorado Springs revealed the occurrence of the lesion in children and young adults who were natives or who had become resident in the area in infancy or early childhood. From this survey, and from data obtained elsewhere, it was established that dental fluorosis was due to the domestic water supplies. A study of the effects of dental fluorosis in inhabitants of an Indian reservation in Oklahoma showed that, though the teeth were badly marred as a result of dental fluorosis, the incidence of caries was markedly reduced. Water in previously-known endemic districts was found to contain fluorine in the quantities of 0·8 p.p.m. to 18 p.p.m.; and, generally, new water supplies provided to eliminate dental fluorosis were free. The use of fluorine-free water was followed by a marked increase in the incidence of caries. Extensive investigations were undertaken to determine the amount of fluorine which would control the incidence of caries without producing mottled enamel. It is fairly well established that the optimum fluorine content of the common water supply would be 1 p.p.m. In June, 1944,

a plan was initiated in two cities to determine the practicability and safety of fluorinating a communal water supply deficient in fluorine, for the purpose of controlling dental caries. Sodium fluoride was fed into the water supply to maintain a fluoric concentration of 0·9 p.p.m. to 1 p.p.m. The experiment will be continued for 10–12 years, during which period periodic dental and physical examination of school children will be made. In addition, research is being undertaken into the effects of topical application of fluorine. *A. Thelwall Jones.*

Tuberculosis in Industry from the Viewpoint of the Physician in Industry. POLLAK, M. (1946). *Occupational Med.*, **1**, 302.

Tuberculosis is the second cause of death among gainfully employed males between the ages of 15 and 64. In a survey in Peoria of workers not under an industrial medical service, 12,129 employees participated voluntarily; the case incidence of active pulmonary tuberculosis was 0·29 per cent. In the Caterpillar Company works, with a medical service, and preplacement, and periodic medical examination, the incidence among 13,547 employees was about half 0·16 per cent. Value of *x*-ray examination of employees has been well shown by the Metropolitan Life Insurance Company, which set up its own sanatorium in 1913, and started yearly *x* rays in 1927. Though the number of its employees grew from 11,530 in 1928 to 14,643 in 1940, the case incidence of clinically significant tuberculosis dropped from 0·49 to 0·09 per cent., and because of lack of an adequate number of patients, the sanatorium closed in 1945. It is the job of the industrial medical officer to spot, and ensure adequate treatment for, the early case of tuberculosis; in this way he can help to eradicate the disease from the community. He must also supervise the patient with arrested tuberculosis and see that he is well rehabilitated and placed in a job within his capacity.
K. M. A. Perry.

A Method for the Determination of Iron in Welding Fume Samples. KEENAN, R. G., and MINDERMAN, B. L. (1946). *J. Industr. Hyg.*, **28**, 32.

The problem at issue was the rapid and accurate analysis of the iron in welding fume samples which contained silica, titanium, and, in some cases, zinc or lead. Many volumetric methods are available; but, as the result of numerous analyses made under controlled conditions, the following method was arrived at for the analysis of the 2,000 samples involved. The fume sample was collected by means of an electric precipitator and dissolved in concentrated hydrochloric acid. The iron was completely reduced to the ferrous state by means of sulphurous acid, the excess of acids being removed by evaporation with sulphuric acid. The iron in the solution was titrated by means of 0·01 normal potassium permanganate at an optimum temperature. Various details of experiment are given in the paper. The accuracy of the method was found to be 98 to 100 per cent. *H. M. Vernon.*

Comparison of the Chronic Toxicity of Triethylene Glycol with that of Diethylene Glycol. FITZHUGH, O. G., and NELSON, A. A. (1946). *J. Industr. Hyg.*, **28**, 40.

Previously published results on the toxicity of diethylene and triethylene glycols have not been consistent, and no long-time study on triethylene glycol has yet been reported. The present investigators used groups of 12 weanling male rats fed on rat biscuits and 1 per cent. cod-liver oil, with drinking water containing 1, 2, and 4 per cent. of the glycols. There was a control group with no glycol. The experiments were continued for two years, and showed that triethylene glycol had no toxic effect. Diethylene glycol retarded the growth of rats, especially when in 4 per cent. strength, but neither glycol had any effect on food consumption. Diethylene glycol produced extensive lesions in the lower urinary tract. With the 4 per cent. glycol, bladder stones composed of calcium oxalate occurred in all but one rat: with the more dilute glycol they occurred in smaller numbers. Bladder tumours, both papillary and intramural, occurred in about half the rats on the 2 and 4 per cent. concentrations. Moderate lesions in the kidneys and liver were produced by the 4 per cent. glycol, and slighter ones by 2 per cent. glycol. Other organs, such as heart, stomach, small intestine, and muscle, showed very slight changes, or none at all.
H. M. Vernon.

Lead Intoxication by Inhalation. COTTER, L. H. (1946). *J. Industr. Hyg.*, **28**, 44.

Poisoning by inhaled lead is still prevalent on the Eastern seaboard of the U.S.A. When, for instance, an elevated railroad is taken down by workmen cutting the steel with blow torches through several layers of paint, the incidence of inhaled lead poisoning may be as high as 80 per cent. unless positive pressure respirators are worn. Some 300 cases were observed, 10 of which are described in some detail. They were mostly middle-aged men who had been exposed from one to three months. They showed abdominal tenderness, cramps, and weakness of muscles. The blood and urine contained only a moderate excess of lead, but the blood showed a significant rise of reticulocytes, and a fall of granulocytes and haemoglobin. The symptoms were so acute, and supervened so rapidly, that the workers were forced to quit before the damage became irremediable. *H. M. Vernon.*

2,4-Dinitrophenol Poisoning: a Case Report. GISCLARD, J. B., and WOODWARD, M. M. (1946). *J. Industr. Hyg.*, **28**, 47.

Many instances are known of poisoning by 2,4-dinitrophenol, which is produced as an intermediate in the manufacture of picric acid. Two fatal cases are here described. In both of them profuse sweating and restlessness appeared after a few months' exposure. After treatment and rest the men returned to their work, but one collapsed and died four hours after he had been taken to hospital, and the other worker died under similar circumstances. The concentration of dinitrophenol dust in the workroom was at least 400 mg. per 10 cubic metres. The factory management then took several steps—such as the installation of exhaust ventilation, strict personal cleanliness of the workers, a complete change of clothing twice a day, and the wearing of protective masks—to remedy the working conditions. An examination of the internal organs of the two victims failed to show the presence of dinitrophenol or of 2-amino-4-nitrophenol, but the urine of the case tested contained 2·08 g. per litre of unchanged 2,4-dinitrophenol, and 50 mg. of the amino derivative. A simple rapid polarographic procedure was used for estimating the dinitrophenol. *H. M. Vernon.*

Atmospheric Concentrations of Fluorides in Aluminium Reduction Plants. YAFFE, C. D. (1946). *J. Industr. Hyg.*, **28**, 29.

Aluminium is produced by an electrolytic process in which a direct current is passed through molten cryolite ($3NaF.AlF_3$) containing alumina. The operation is carried out in electrolytic cells, or 'pots.' The pot is a steel box, about 8 ft. by 4 ft. and 2 ft. deep. It is lined with carbon, and the anode consists of one or more carbons suspended in the molten bath. A number of pots are connected in series, and the passage of a current of 8,000 to 40,000 amperes causes metallic aluminium to be deposited on the bottom of the pot, which serves as the cathode. The carbon dioxide liberated carries off fumes containing fluorine compounds, including some HF. The HF and the fluoride salts were estimated separately by passing the air sampled through an electrostatic precipitator to remove particulate matter, and then through an absorbent solution of 1 per cent. sodium hydroxide in an impinger in order to collect HF. The men especially exposed to the fumes were the pot men,

tappers and carbon crews; and air samples collected in the places where they worked contained from 1·8 mg. to 58·4 mg. of fluoride per 10 cubic metres. The fluorine was almost equally divided between the precipitator and the impinger samples, and there were no significant differences in the degree of exposure of the various groups of men, the averages being from 8 to 10 mg. of F per 10 cubic metres. The allowable limit of HF is generally considered to be 3 parts per million or 24 mg. per 10 cubic metres, so the men seldom ran any risk of poisoning. *H. M. Vernon.*

Nitrogen Dioxide—Rate of Oxidation of Nitric Oxide and its Bearing on the Nitrogen Dioxide Content of Electric Arc Fumes. ELKINS, H. B. (1946). *J. Industr. Hyg.*, **28,** 37.

Nitric oxide is produced in the electric arc; its rapid oxidation to nitrogen dioxide is, however, generally assumed. On the other hand, Bodenstein concluded that under the usual conditions most of the nitrogen fixed by the air would remain as the monoxide for some time. Direct observations by various investigators showed that the ratio of NO_2 to NO was as 1 to 150, as 1 to 20 and as 1 to 6. The NO formed in the arc is not converted into NO_2 until it has cooled, and from data obtained by other investigators Elkins makes the following calculations: (1) If the welding arc is producing 0·01 cubic feet per minute of NO in a 1,000 cubic-foot room which is exhausted at the rate of 100 cubic feet per minute (one air change in 10 minutes), the average NO_2 will be 22 parts per million. (2) If the ventilation is increased to 200 cubic feet per minute the NO concentration will be only 3·5 parts per million, or a sixth as much. (3) Conversely, if the ventilation is halved, the NO_2 concentration will be no less than 104 parts per million. Since NO, though it has certain toxic effects, is not an irritant gas like NO_2, good ventilation is evidently of great importance. The maximum allowable concentration of oxides of nitrogen is considered to range from 10 to 25 parts per million. *H. M. Vernon.*

Chromates in Animal Nutrition. GROSS, W. G., and HELLER, V. G. (1946). *J. Industr. Hyg.*, **28,** 52.

There is an increasing use of chromate salts and of chromic acid in plating baths, and several studies on the toxicity of chromates have been reported. The present investigation was made in order to ascertain the level of chromate salts that an animal can ingest without impairment of general health and reproductive powers. The rate of growth of growing mice and rats over periods of two or more months was studied, and it appeared that for white rats the maximum non-toxic level of potassium chromate in the drinking water is 500 parts per million. For mature white rats and mice the maximum non-toxic level of zinc chromate in the food is 1 per cent., but young rats are stunted and made sterile by 0·125 per cent. of this chromate. For rats of all ages, 0·125 per cent. of potassium chromate is the maximum amount which can be added to the food without deleterious effect. Digestion trials made on three mature female rabbits showed that drinking water containing up to 500 parts per million of potassium chromate did not affect the utilization of food, but that 1 per cent. of zinc chromate in the food markedly lowered the digestion coefficients of practically all its components. Tests for chromium salts in the blood and urine were negative, so presumably they do not enter the blood stream from the alimentary canal. *H. M. Vernon.*

The Arterial Lesions of Silicotic Blocks. CROZIER, L., ODE, L., and ROCHE, L. (1945). *Presse méd.*, **53,** 638.

Autopsies on miners who have died from silicosis often disclose 'blocks' or hard fibrotic masses of varying size. These occupy part or the whole of a lobe. Histological examination of these lesions reveals marked changes in the arteries of the affected areas. Many of them are practically avascular, as shown by arterio-pneumography carried out post mortem. The technique was an easy one. After removal of the lungs a radio-opaque substance was injected into the pulmonary arteries, and the chest *x* rayed. A dilute solution of baryta was used. Three cases were investigated.

The first patient had worked all his life as a tunneller. He was very dyspnoeic when admitted to hospital, and a radiograph showed typical silicotic changes. There were bilateral diffuse changes of miliary type, and under the right clavicle a mass resembling a tumour There was no clinical or laboratory evidence of associated tubercle. The heart was failing gradually over a period of six months, and the film showed cardiac enlargement. Dropsy developed. Arteriography performed post mortem revealed the almost complete avascularity of the affected zone, where shadowing indicated the position of the lesion. The large vessels were narrowed and often filiform, the arterioles were almost invisible, and there were scattered areas of several square centimetres which were completely avascular. This was the most typical picture obtained.

The second subject, a rock miner, was also very dyspnoeic when admitted to hospital. In his case radiography confirmed the diagnosis of silicosis, and a silicotic block was seen in each lung in the middle zone, with more diffuse lesions in other parts. This man died of heart failure with anasarca. Radiography after injection of the arteries showed marked vascular changes, especially in the right lung. The third patient had no radiograph before death, but the arteriography was similar to the other two.

Policard has pointed out that the fibrosis of pneumoconiosis begins round the vessels, and that a sleeve of fibrous tissue soon envelops these, together with the lymphatics and the bronchi. The arteries resist the longest, but in the silicotic blocks vascular lesions are constant, both in those fully formed and in those still developing. Vessels are obliterated both by thrombosis and by swelling of the elastic tissue. Not only are the medium-sized arteries involved, but also the smaller ones accompanying the bronchioles. Destruction is eventually complete, leaving a mass of sclerotic tissue. The part played by tuberculosis is a matter for discussion, but vascular lesions are more common when there is a marked tuberculous factor. Vascular changes are greatest in caseating areas. The cardiac embarrassment due to the existence of these large, dense lesions can be readily understood. Nevertheless, with a normally beating heart, the rate of blood flow through the lungs is usually normal; though one will admit that the flow through the narrowed loops is more rapid than previously. The blocks often exhibit degenerative changes with cavity formation and the appearance of copious purulent sputum, usually dark in colour. No doubt associated tuberculosis is responsible in many cases, and Croizier and E. Martin nearly always found tuberculosis in this black sputum. But it is likely that in some cases cavity formation is due to mortification from ischaemia. Schlaepfer showed that ligature of branches of the pulmonary artery was quickly followed by the development of sclerotic areas, and it is likely that arterial lesions in silicosis, with associated damage to veins, lymphatics and bronchi, play a part in the rapid development of the lesions described. *G. C. Pether.*

The Natural Ventilation of Unheated 'Closed Rooms.' CARNE, J. B. (1946). *J. Hyg. Camb.*, **44,** 314.

Despite the importance of the subject, there is a deficiency of scientific information about the natural ventilation of rooms in dwelling houses. Two rooms, one facing west and one facing east, in a house in No. 699 Old Kent Road, each with a fireplace and ventilation wall grating (50 sq. in.) were examined with the doors and windows closed to determine the natural air change under different climatic conditions from Nov., 1938, to March, 1939. Air change was determined by introducing CO_2 into the air of the room and estimating the concentration before and after test periods. Air

influx increased rapidly in proportion to the size of the chimney flue until an area of 15 to 20 sq. in. had been provided; thereafter the influx increased slowly. Maximum ventilation through the wall grating occurred when the wind was normal to the wall; the higher the velocity of the wind, the wider the angle through which the wind might veer and still maintain a high influx.

Air blown against a model house creates high pressure against the windward wall, low pressure on the leeward side, and a zone of turbulence at the junction of the roof and leeward wall. There is a negative pressure exerted up the flue. The influx into the room is determined by the pressure difference between the room and the centre of the house and by the size of the openings from the room to the centre of the house, rather than by the size of the flue or wall grating. The pressure in the centre of the house will be the sum of the negative pressure due to the flue effect in the other rooms and the wind pressure (negative or positive) against the side of the house. To ensure maximum natural ventilation of a room, the flue should terminate outside the region of turbulence and in the region of negative pressure, the room should have an opening to the centre of the house, and there should be an opening between the centre of the house and each of two opposite walls.

T. A. Lloyd Davies.

Injury Resulting from Use of Aerosol Bomb. METZLER, D. F. (1946). *Publ. Hlth. Reps. Wash.*, **61,** 546.

Failure in the mechanism of an aerosol bomb allowed a mixture of pyrethrum, DDT, and Freon to escape on to the hand of an operator attempting to carry the bomb out of a building. When he dropped the bomb his hand was frozen in the shape in which it had been cupped over the bomb. An injury equivalent to a third degree burn was sustained, but analysis of the blood indicated no toxic effect from the DDT. *T. A. Lloyd Davies.*

Industry and Its Environment—Physical and Social. LORD FORRESTER (1945). *J. roy. Inst. Publ. Hlth.*, **8,** 306.

An industrialist makes a strong plea that managements should plan work to fit human beings and that industry should no longer defile the countryside with smoke and scrap heaps. If modern processes are used, he says, factories need no longer be ugly. There must be co-operation between industry on the one hand, and town and country planning authorities on the other; and, so that the citizen may take full advantage of the new surroundings, educational and health services must be fully developed. *T. A. Lloyd Davies.*

Some Methods of Measuring Ventilation. LIDWELL, O. M., and LOVELOCK, J. E. (1946). *J. Hyg. Camb.*, **44,** 326.

The determination of ventilation of a room by measuring the amount of air entering and leaving is difficult: the alternative method is to estimate the concentration of a tracer substance. If the air is completely and continually mixed, the rate of disappearance is:

$$\frac{\dot{v}}{V} = \frac{2{\cdot}30}{t}\ (\log_{10} C_0 - \log_{10} C)$$

where c is the concentration of tracer substances remaining after time t, v is volume of air entering and leaving a room of volume V. Trace substances must be stable, odourless, and non-toxic. Apparatus and methods of measuring the concentration of tracer substances are by (1) ultra-violet absorption, (2) absorption of vapour and colorimetric estimation, either visually or photo-electrically, (3) development of colour on a treated test paper. Acetyl acetone and ethyl aceto-acetate fulfil the requirements for tracer substances for (1), tertiary butyl hypochlorite for (2) and (3). *T. A. Lloyd Davies.*

The Relationships Between Carbon Monoxide, Oxygen, and Hemoglobin in the Blood of Man at Altitude. LILIENTHAL, J. L., RILEY, R. L., PROEMMEL, D. D. and FRANKE, R. E. (1946). *Amer. J. Physiol.*, **145,** 351.

To determine the permissible concentration of CO at various pressure altitudes, three men (a heavy smoker, a light smoker, and a non-smoker) were exposed to a mixture of CO, O_2 and N_2, at varying pressures, until CO in the blood reached equilibrium. The amount of COHb produced when a man is in equilibrium with CO air mixture may be calculated from a modification of Haldane formula as follows:

$$(\text{COHb}) = \frac{\text{MpCO}}{\text{pO}_2 + \text{MpCO}}$$

M = relative affinity constant of Hb for CO compared to O_2.

$pO_2 + MpCO$ = total effective gas tension may be read from standard table.

pO_2 = average alveolar tension at pressure altitude.

In these experiments, M was found to be 204±10 per cent. (and does not differ in smokers and non-smokers). Symptoms produced by CO are proportional to the concentration of COHb in the blood and duration of exposure. *T. A. Lloyd Davies.*

The Effect of Low Concentrations of Carboxyhemoglobin on the 'Altitude Tolerance' of Man. LILIENTHAL, J. L., and FUGITT, C. H. (1946). *Amer. J. Physiol.*, **145,** 359.

Difficulty in determining the effect of the absorption of CO is experienced because, depending on the symptoms or the delicacy of the function chosen as a test to indicate toxicity, different COHb concentrations will be assigned as the significant level. Experience has shown that men can fly safely at altitudes of 8,000–10,000 ft. without additional oxygen and by chance deterioration in the flicker fusion frequency test, because of anoxia, occurs at this altitude pressure. The flicker fusion frequency forms a useful test of the effect of small increases in COHb in lowering the 'ceiling' of flight or the height at which additional O_2 is required. At 10,000 ft. air containing 0·008 per cent. CO will convert 10 per cent. of Hb into COHb. With this concentration of COHb, unequivocable impairment of visual discrimination and flicker fushion frequency will occur. Rigid standards for permissible limit of CO in the air must be enforced.

T. A. Lloyd Davies.

The Effect of Oxygen Pressure on the Uptake of Carbon Monoxide by Man at Sea Level and at Altitude. LILIENTHAL, J. L., and PINE, M. B. (1946). *Amer. J. Physiol.*, **145,** 346.

The design of war craft may prevent the complete exclusion of carbon monoxide; in aircraft the absorption of CO at high altitudes assumes importance. Eight men and one woman were exposed to mixtures of O_2 and CO at varying pressures until the CO content of the blood showed stability had been reached (at a level not exceeding one quarter of anticipated equilibrium of CO intake value). At sea level, symptoms due to CO did not occur until COHb exceeded 15 per cent.; the uptake was a linear function of the CO exposure index (=partial pressure CO (mm. Hg) in inspired air, times minute respiratory volume, times duration of exposure in minutes), times 0·05; at pressure altitudes of 15,000 to 25,000 ft., O_2 was added to the inspired mixture to produce O_2 pressure equivalent to sea level; the CO absorption is inversely proportional to the partial pressure of O_2. The total barometer pressure has no apparent influence on the uptake of CO: the effects of minimal altitude anoxia added to the effect of circulating COHb combine to produce moderately severe symptoms.

T. A. Lloyd Davies.

Lumbar Herniations of Intervertebral Discs—Value of Surgical Removal for Naval Personnel. WHITE, J. C. and PETERSON, T. H. (1946). *Occupat. Med.*, **1**, 145.

The authors report a series of 46 cases among Naval personnel operated upon for herniations of lumbar intervertebral discs. Seventy-five per cent. of cases were restored to full or limited duty, a percentage comparing favourably with an average of 68 per cent. of good to excellent results reported in 12 civilian series totalling 938 patients. While admitting grounds for the gloom in prognosis shown by the Office of the Surgeon General, U.S. Army, and by Air Vice-Marshal Symonds, speaking of Royal Air Force personnel, the authors believe their series illustrates the usefulness in the Services of operation for this condition, provided the cases are carefully selected. Their criteria were (1) severe low back pain and sciatica, (2) condition unrelieved by at least one month's conservative treatment, (3) positive filling defects in the Pantopaque spinogram and (4) that patients be otherwise physically fit. Of 101 patients examined, only 46 were subjected to operation, all but two of whom presented positive filling defects. These two cases were operated upon on strong clinical grounds, and both proved to have herniated discs. One negative result on exploration, and the finding of other causes of root compression in six cases are reported in the series. Of 24 of these cases followed up for up to 2½ years, 54% returned to unlimited duty, 21% to limited duty, 21% were still of uncertain status in the service, and 4% were invalided.

With regard to carrying out Spinal Fusions at the same time as the removal of the herniated disc. The authors feel that, where structural bony and ligamentous defects are obvious at the time of operation, spinal fusions should be carried out at the same time as the removal of the herniated disc. But, bearing in mind the youth and good physique in general of services cases, and the common loss of desire to return to full duty after a prolonged period on the sick list, they prefer to rely on the healing of minor ligamentous injuries during convalescence, and their convalescent regime is directed to the earliest possible resumption of normal everyday activity in order to minimize the importance and severity of the operation in the patient's mind. Spinal fusion, therefore, is reserved as a secondary procedure for those cases that relapse on resumption of activity, except where the need for it is quite obvious at the primary operation. *O. J. Vaughan-Jackson.*

The Acute Toxicity of DDT following Intravenous Injection in Mammals. PHILLIPS, F. S., and GILMAN, A. (1946). *J. Pharm. exp. Therap.*, **86**, 213.

Acute DDT intoxication was studied in cats, dogs, monkeys and rabbits by the method of intravenous injections of an emulsion of DDT. Its toxicity in this form was found to be of the same order for each mammal, but, when compared with the oral route, the lethal dose of intravenous DDT was 10 times less, and resultant symptoms appeared more rapidly, 5 minutes after injection and lasted a significantly shorter time. The symptoms produced were muscle tremors and convulsions, tonic and clonic, followed usually by a period of central depression. There were no residual toxic effects from single large doses of DDT, such as the degenerative changes that characterize chronic DDT poisoning, which suggested that such symptoms were caused by DDT itself rather than by a metabolic transformation product. With higher doses death occurred, either from cerebral depression and respiratory failure directly following the cerebral excitation, or from ventricular fibrillation (dogs). A limited number of anti-convulsant and central depressant drugs were investigated to determine an effective method of treating acute DDT poisoning. Of the various therapeutic agents studied, phenobarbitone was found to be outstanding in that it controlled the characteristic tremors and convulsions produced by DDT in doses which caused a minimal degree of central depression. *J. L. Lovibond.*

The Excretion of DDT (2,2–Bis–(P–chlorophenyl)–1,1,1–Trichloroethane) in Man, together with Clinical Observations. NEAL, P. A., SWEENEY, T. R., SPICER, S. S., and VAN OETTINGEN, W. F. (1946). *Publ. Hlth. Reps., Wash.*, **61**, 403.

In rabbits, DDT administered in olive oil by mouth is partially excreted as di-(p-chlorophenyl) acetic acid (DDA). A healthy male volunteered as an experimental subject. In July, 1943, he was exposed to 1 mg. DDT per 1,000 cubic feet of air for 1 hour daily for 6 days, and four weeks later to a higher concentration for 1 hour daily for 5 days; in December, 1944, the same subject ate 500 mg. in olive oil without untoward effect. In September, 1945, he ingested 770 mg. (11 mg. per kilo bodyweight) of recrystallized DDT in 25 c.cm. olive oil on an empty stomach. There was no evidence of liver damage, and the blood sugar remained normal. All functions of the C.N.S. were normal. Following the ingestion of DDT, there was a sharp rise in the urinary excretion of DDA, reaching a maximum (4,000 mg.) on the second day. Excretion decreased rapidly on the third and fourth day, and then gradually fell during the subsequent 10 days. DDT did not appear in the urine.
T. A. Lloyd Davies.

Influence of Iron Salts on the Toxicity of Lead. HEPPEL, L. A., and KORNBERG, A. (1946). *Proc. Soc. exp. Biol. Med.*, **61**, 220.

The action of calcium and phosphorus in modifying the effects of lead has been closely studied, but the administration of iron prevents the loss of weight and anaemia occurring in rats fed on diets containing lead acetate. Male weanling rats on a diet containing 0·03% of lead (and after 31 days on lead, 0·09%) remained constant in weight after intervals of 65 to 75 days, with a constant or slight increase in Hb in the blood if 1·13% of ferric citrate was added to the diet. Rats on the same diet without ferric citrate lost weight and became anaemic (Hb reduced from 15 gm. per 100 c.c.m to 9·9 gm. per 100 c.cm.). Sodium citrate was ineffective. The addition of 0·25% of ferric chloride to the diet failed to prevent loss of weight or anaemia, and 0·2% of ferrous sulphate added to the diet prevented anaemia but not loss of weight. Experiments in progress are stated to indicate that rats fed on iron supplement show a lower lead content of the tissues: a possible explanation is that iron interferes with the absorption of lead.
T. A. Lloyd Davies.

Streptomycin in Experimental Plague. HORNIBROOK, J. W. (1946). *Publ. Hlth. Reps., Wash.*, **61**, 535.

Streptomycin is isolated from an organism resembling *Actinomyces griseus* and has a low toxicity (L.D. 0=35 mg. and L.D. 100_4=135 mg. per 20 g. mouse of preparation containing 30,000 units per gramme). Mice weighing about 18 g. were inoculated subcutaneously with up to 6,000 organisms of *P. pestis*, and streptomycin was inoculated intraperitoneally at various times. Controls were given normal saline. Visible swelling at the site of inoculation was considered to be one of the signs of infection: animals not showing swelling at the time of treatment were excluded. Streptomycin was given half an hour before and 24 hours after infection. A total dose of 2 mg. of streptomycin protected all animals from death, whether given in two or three doses. Sixty-six per cent. of animals infected for 72 hours before treatment with 2 mg. of streptomycin in two doses at a 24-hour intervals died in 14 days. In a group of animals infected for 48 hours before treatment, 10% died within 14 days after 2·0 mg. streptomycin; 63% died after treatment with 2 mg. sodium sulphadiazine, compared with a death rate of 88% in the controls. *T. A. Lloyd Davies.*

Life Tables for Social Classes in England. TIETZE, C. (1943). *Milbank Memorial Fund Quarterly, New York*, **21**, 182.

William Farr's pioneer accomplishment in tabulating male deaths by occupation has been steadily improved at

each subsequent census. The 1931 tables (Registrar General's Decennial Supplement, 1931, England and Wales II*a*, *Occupational Mortality*) are of more lasting interest than usual because, unless the date of the next census is advanced, new tables will not be available until some years after 1951. To calculate the percentage distribution of social classes, the following deviations from the Registrar General's tables were made: (1) farmers, gardeners, agricultural labourers, and (2) miners were removed from social class II, III, IV, and V as both these groups are homogeneous and sufficiently large to influence the total mortality of the class (miners have high and farmers, etc., low mortality); and (3) 700,000 clerks were transferred from class III to II to join their black-coated fellows (employers and managers), into whose ranks many will ultimately advance.

The percentage distribution of occupied males is as follows:

Social Class	Males 16 and over	Per cent.
I. (Professional, etc.) ..	335,182	2·5
II. (Intermediate between I and III)*	2,211,533	16·2
III. (Skilled workers)	5,313,129	38·9
IV. (Intermediate between III and V)	1,560,583	11·4
V. (Unskilled workers) ..	2,239,738	16·4
Farmers, gardeners, agricultural labourers ..	1,117,281	8·2
Coal miners	868,523	6·4
Total ..	13,645,969	100·0

* Almost 90% of Class II are employers and managers in business and clerical workers.

To calculate life tables, adjustments were made for the mortality of illegitimate infants according to the father's occupation (Registrar General records mother's occupation), for the mortality between 2 years and 16 years, and for the failure to return to the occupation of retired males in the higher age groups. To avoid the appearance of pseudo-accuracy, a radix of 1,000 was used, and expectation was shown to one decimal point.

Age	I	II	III	IV	V			
	Professional, etc.	Intermediate between I and III	Skilled workers	Intermediate between III and V	Unskilled workers	Farmers, etc.	Coal miners	All males
	Survivors at Exact Ages							
0	1,000	1,000	1,000	1,000	1,000	1,000	1,000	100,000
1	961	947	935	924	910	938	905	92,814
2	956	941	924	910	890	930	886	91,394
16	930	913	893	877	854	900	850	88,186
20	916	904	885	868	845	894	838	87,245
25	901	891	871	854	830	882	822	85,824
35	876	865	843	824	799	858	789	82,885
45	838	822	800	771	747	824	739	78,357
55	759	738	719	677	654	762	658	70,041
65	605	576	568	520	503	641	518	54,899
70	484	450	451	402	391	538	400	43,361
75	349	303	312	264	258	402	264	29,665
	Complete Expectation of Life at Birth							
0	63·1	60·8	60·0	57·3	55·7	63·0	55·7	58·74

The difference between maximum and minimum values is 7·4 years: this is little more than the increase in expectation of life observed in England as a whole between 1911 and 1931. The expectation for Class I and farmers, etc., is virtually the same; in youth the number of survivors in Class I is higher, but in later years farmers have the advantage. Well educated and well-to-do parents are more successful in preventing deaths among their children, but farmers either come of a sturdier stock or their mode of life is healthier. In interpreting Table II, caution is necessary as life table technique has been applied to social classes which are not closed populations. *T. A. Lloyd Davies.*

Medical Progress: Industrial Hygiene. TABERSHAW, I. R. (1945). *New Engl. J. Med.*, **233**, 437.

This is a short review of recent advances in occupational medicine. Radioactive substances may not, after all, be the cause of the lung cancer found in Schneeberg and Joachimsthal, as arsenic dust, chronic lung diseases, and hereditary susceptibility are now suspect. The toxicity of quartz may be due to the colloidal silica liberated on the quartz particles by tissue fluids. Perfused colloidal silica has a broncho-constrictor effect. 'Eggshell' calcifications have been found in the hila and sometimes in the parenchyma of the lungs in slate quartz miners; some also were found in people who were exposed equally to silica and limestone. Beryllium can cause a pneumonitis and has been under investigation. Routine radiographs of the lungs of farmers have revealed symptomless calcifications thought to be due to an *Aspergillus*. Recent work in the cotton fields suggests that byssinosis does not exist there, but that an acute illness called 'cotton fever' may attack the new worker, but has no sequelae.

The use of powdered metallic aluminium or hydrated alumina (XH 1010) for therapy and prophylaxis in silicosis is now widespread, but with varying results; the choice of which to use seems to depend on individual preference. Control of the dust in the atmosphere remains the most important prophylactic measure.

Poisoning due to lead and benzene is less frequent. Carbon monoxide is still the most frequent cause of industrial intoxication. Apart from better-known industrial poisons, toxic effects from methyl bromide and ethylene chlorhydrin are reported. Study of the physical conditions of working shows that wet-bulb thermometer readings below 91° F. are tolerable, but at 94° F. or above sustained effort is impossible, and cases of heat exhaustion occur. Thiamine and choline requirements of the body rise with heat.

It has been found that sodium citrate assists the excretion of uranium salts. The luminous-dial-painting industry has expanded, but the known effective countermeasures to chronic radium poisoning have not been fully applied in all places. In half the cases of industrial dermatitis, no single agent can be incriminated. There is a phenomenon in dermatitis where a sensitive subject can be made progressively less allergic to a substance by repeated exposure. This 'hardening' does not occur in all persons. *J. N. Agate.*

Contact Dermatitis of Eyelids Caused by an Anti-oxidant in Rubber Fillers of Eyelash Curlers. CURTIS, G. H. (1945). *Arch. Derm. Syph., Chicago*, **52**, 262.

Seven cases are described of contact dermatitis after the use of eyelash curlers, part of the frame of which had a rubber filling or band.

Four points are given as diagnostic. (1) Eruption is subacute. (2) There is a linear appearance. (3) On the upper lid a narrow band of normal skin is left along the tarsal fold or between the dermatitis and lid margin. (4) Eyelashes show artificial curling. Patch tests with rubber filling were positive in all cases. In five cases the sensitizing factor was shown to be the anti-oxidant 1% phenyl-beta-naphthylamine, and in the remaining two this factor was strongly suspected.

Geoffrey A. Hodgson.

Allergic Aniline Dermatitis: an Occupational Accident. ZANCHI, A. F., and BONDUEL, A.A. (1945). *Revista Asoc. med. Argent.*, **59**, 1322.

The patient had worked many years with aniline dyes used for textiles. He used blocks which were dipped in the various colours before being applied to the material. Thus his hands were often in dye and he inhaled the fumes. The first changes were seen in the hands, which exhibited reddened areas with vesicles and much serous discharge. The spaces between the fingers suffered most, being painful and burning. Eczema was diagnosed, and he stopped work for two weeks. Work was resumed, but the rash returned and spread to the face, forearms, and feet. He also had digestive symptoms, diarrhoea, nausea, and severe headache. He then stopped work for four months, but on resuming was affected again with an extensive dermatitis and had then to abandon his employment for good.

Clinical examination, a blood count, and stool examination for parasites revealed no abnormality.

Patch tests were done with cotton impregnated with three solutions as follows: (1) aniline 50 g., NaOH 10 g., Turkey red oil 30 g., alcohol 30 g., neutral chromate solution 50 g.; (2) aniline 5%, Turkey red oil 3%, alcohol 2%, caustic soda 3%; (3) yellow wood oil 30%, campeche and bisulphite 2%, formic acid 10%. After 48 hours the patches were removed, which showed red areas in tests 1 and 2. These were surrounded by a slightly painful inflammatory area. Test 3 was negative. On the third day the affected areas showed more marked changes and the workman felt ill, developing diarrhoea and then bloody stools. The test was repeated eight days later, with even more marked and similar results. This time the face was swollen, the eyes puffy, and there was worse diarrhoea with insomnia.

The writers consider this a certain case of occupational poisoning. They point out that most aniline dyes are toxic only when they contain pure aniline or impurities such as arsenic, lead, and mercury. Absorption is mostly by the lungs and rarely by the gut. But dyes which have condensed on material may penetrate through the skin, especially if the temperature is over 30° C. It combines with haemoglobin to form methaemoglobin, and changes or destroys the red cells. Oxidation of aniline in the blood produces a cyanotic tint. *G. C. Pether.*

Nitrous Fume Poisoning in Welders. HATT, F. (1946). *Schweiz. med. Wschr.*, **76**, 483.

The author describes the illness and death of a workman who worked inside a boiler of about 10 cubic metres volume. It had a manhole through which he entered and left at intervals. He was inside for a total period of 25 minutes, and he sweated heavily, coughed, and had a very red face. As he complained of the heat and smell, air was blown in during an interval and blue fumes were driven out. After this the patient did three more hours' welding in the open, and when his work was over he had some tightness in the chest. He was, nevertheless, able to cycle home part of the way, then take train, and then walk a short distance. He usually covered this in ten minutes but required an hour to do so and could scarcely get into his house. In the night his headache and dyspnoea were so severe that he called for help. Next morning he was seen by a doctor and sent to hospital. On admission there was marked orthopnoea and cyanosis and the patient was incoherent. There was extensive oedema of the lungs. Venesection was performed and 260 c.cm. of blood was removed. Morphia and strophanthin were administered. The blood exhibited a leucocytosis; the Takata reaction was negative. The general condition got worse, and oxygen was given continuously, as also intravenous hypertonic glucose and strophanthin. The patient died in coma. After death 3·88% methaemoglobin was found. Hb. was 109%. At necropsy there was evidence of death from oedema of the lungs and asphyxia attributed to poisoning with nitrous fumes while working as an acetylene welder. The author says that oxides of nitrogen may also be formed when the electric arc is used for welding, and precautions are needed, especially for those working in confined spaces. The long latent period before the development of symptoms is typical of the condition. *G. C. Pether.*

The Problem of Carbon Monoxide Poisoning from Coal Gas. MOESCHLIN, S. (1946). *Schweiz. med. Wschr.*, **76**, 508.

The writer says there has been an increase in CO poisoning, not only from charcoal, but also from coal gas. In Switzerland in 1939 there was 8% of CO in the coal gas, but it rose to 25% in 1944. Deaths by suicide and by accident have doubled in the decade ending 1944. The writer suggests that many slight cases of poisoning are not reported, and that repeated exposure may cause insidious symptoms. He stresses the defects of memory and of perception which may occur with acute poisoning, the development of Korsakoff's syndrome, and the chance of damage to the mid-brain and the basal ganglia. Athetotic and choreiform symptoms have been noted. Disorders of sleep are known, and respond very badly to the usual sedatives. Graves' disease has been reported, attributed to injury to the diencephalon. Peripheral neuritis and myocardial damage is also known. The writer inquires whether these changes are due merely to anoxaemia or to some specific toxic effect. He thinks the myocardial damage derives from anoxaemia and points out that some think the heart damage of heavy smokers is due to CO rather than to nicotine. Cerebral damage to a permanent nature may also arise from anoxaemia. CO may be a heavy-metal poison in some circumstances, hence its affinity for the iron-containing haemoglobin. Tissue cultures do not seem to have their growth inhibited by CO, but this is no proof that the gas is harmless to nerve cells. The writer suggests that CO may do some damage by inhibiting the action of certain enzymes which contain heavy metals, this being a specific toxic effect. Experiments in guinea-pigs have shown a hypertrophy of the thyroid with increased metabolic rate. This may be secondary to some changes in the brain. Glycosuria and a rise of blood sugar occur in cases of CO poisoning. Many Swiss writers think that Parkinsonism can often be traced to chronic CO poisoning. Vertigo, headache, and insomnia are common; defects of memory are typical. Fine tremors and ataxy occur, and such 'neurasthenic' symptoms as loss of energy and disinclination to work.

Although in theory methylene blue may relieve CO poisoning, the degree of relief thus obtained is small. Some writers advise the injection of thionin solution, as this accelerates the dissociation of CO. On the whole the most effective procedure is to administer from 6 to 10% CO_2 in oxygen. If the patient is still unconscious after 2 to 3 hours, venesection is advised. Coramine may be used as a respiratory stimulant. For convalescents a stay in the mountains is valuable.

G. C. Pether.

Dust Control in Haematite Mining. I. The Effect of Wet Methods. TEBBENS, B. D., and TABERSHAW, I. R. (1946). *J. industr. Hyg.*, **28**, 100.

Dust concentrations in a group of haematite mines were investigated before and after instituting a reduction of dust by water jets. The haematite ore was in a seam 8 to 14 feet thick, and it contain 15 to 20% of free silica, while settled dust samples contain 9 to 16%. In the drilling operations the hollow steel drills used discharged 2 or 3 gallons of water per minute. The broken rock blasted during the previous shift was thoroughly wetted by sprinkling with hoses, while main haulage ways and headings were wetted two or three times a week by means of tank cars. The dust was determined by the use of a midget impinger, and the average dust concentration in million particles per cubic foot of air was reduced in the drilling operation from 151 to 22, in loading from 107 to 23, and in ore

transportation (in headings and haulages) from 70 to 8. Ventilation was the same before and after the introduction of water, and psychrometric observations showed that the water did not appreciably affect the relative humidity of the air or the average environmental conditions relating to comfort. It was considered that in the group of mines investigated it was desirable to maintain the dust concentration at less than 25 million particles per cubic foot. Of the 125 dust samples analysed before the use of water, only 13% attained this standard, whilst of the 109 samples analysed after it, 69% attained it. *H. M. Vernon.*

Evaluation of Urinary Lead Determinations. LEVINE, L., and FAHY, J. P. (1946). *J. industr. Hyg.*, **28,** 98.

The authors record the frequency distribution of urinary lead concentrations of 1,156 samples of urine taken from exposed workers. The curve rises sharply to a maximum of about 0·10 mg. of lead per litre, declines sharply at concentrations between 0·10 and 0·30 mg., and then falls more slowly to 0·60 mg. Only 1·1% of the samples contained more than 0·60 mg. of lead, the highest being 0·86 mg. per litre. Some of the persons in the upper marginal limits of lead excretion received hospital treatment for plumbism, and in many cases such treatment was likewise given at much lower levels. The level of urinary lead excretion usually accepted as indicative of hazardous exposure is 0·20 mg. per litre. This value was exceeded by 24% of the samples. *H. M. Vernon.*

An Experimental Evaluation of Methionine in the Therapy of Liver Injury from Carbon Tetrachloride. SHAFFER, C. B., CARPENTER, C. P., and MOSES, C. (1946). *J. industr. Hyg.*, **28,** 87.

Recent investigations have shown that depletion of the protein store from the body renders the liver more susceptible to the toxic action of poisons such as chlorinated hydrocarbons, the severity of the damage being roughly parallel to the extent of the depletion. If animals are fed with protein immediately before the liver poison is administered the severity of the damage is reduced, this reduction being dependent on the sulphur-containing amino acids of the protein, viz. methionine and cystine. It is stated that when methionine is given as long as four hours after chloroform anaesthesia, it will protect a protein-depleted dog from fatal liver injury. Also it is stated that methionine has a beneficial action on acute carbon tetrachloride poisoning in man. One investigator obtained favourable results in cases of toxic hepatitis caused by trinitrotoluene, but a group of five investigators found no significant effect. The present inquiry was undertaken to determine what value, if any, methionine possesses in the treatment of toxic hepatitis induced by carbon tetrachloride. Liver damage was produced in 28 dogs by the intraportal injection of undiluted carbon tetrachloride in dosages ranging from 0·05 to 0·252 ml. per kg. Fifteen of the animals received 0·3 g. per kg. of methionine post-operatively, and, in some cases, a second dose of equal size 18 hours later. Comparison of the animals with controls given glycine or saline failed to demonstrate any consistently beneficial action of the methionine in the prevention or repair of the liver damage. This conclusion is based on the length of survival, estimations of serum phosphatase, urinary pigments, etc., and on the gross and histopathology. *H. M. Vernon.*

Eight Cases of Methyl Chloride Poisoning with Three Deaths. MCNALLY, W. D. (1946). *J. industr. Hyg.*, **28,** 94.

Poisoning by the refrigerant, methyl chloride, does not occur so frequently now as it did in 1928 and 1929, because of the improved construction of electric ice boxes and the addition of the warning agent, acrolein. However, this agent is not absolutely dependable, in spite of its pungent odour, for persons with a defective sense of smell or those soundly asleep do not always notice it. In one case described in this report, two persons living in an apartment hotel were not seen for four days, and when the apartment was opened they were found dead, lying on the floor. There was a smell of acrolein, and a leaky connexion was found in the ice box. The other cases described in this report were likewise due to leakages. In all the subjects there was a rise of temperature, increased pulse and lowering of blood pressure. There was a primary anaemia with mild leucocytosis. The pupils were widely dilated and reacted sluggishly to light. Anuria lasted 48 hours. The therapy consisted in oxygen inhalations; also of continuous intravenous injection of Hartman's solution and 5% glucose, and of intramuscular injection of liver and iron. Alkalinization of the patients is important. *H. M. Vernon.*

Summary of Twenty-Two Trinitrotoluene Fatalities in World War II. MCCONNELL, W. J., and FLINN, R. H. (1946). *J. industr. Hyg.*, **28,** 76.

In the first world war there were, in the United States, about 17,000 cases of and 475 deaths from TNT poisoning within a period of 7½ months. In the present war only 22 fatalities were recorded during the period June 1, 1941, to Sept. 1,1945, in spite of the fact that many times the number of workers were employed. Of the fatal cases, 8 died of toxic hepatitis, 13 of aplastic anaemia, and one from a combination of the two. Only a third of those who died were exposed to an average concentration of TNT over the generally accepted maximum allowable concentration of 1·5 mg. per cubic metre of air, but it is not known how much TNT was ingested or absorbed through the skin. Hepatitis occurred more frequently among the younger age group of workers, averaging 30 years, and aplastic anaemia among the older group, averaging 45 years. The former group had a much shorter period of exposure, with a median of 63 days, as compared with a median of 216 days for the aplastic anaemia cases. No single test of TNT poisoning has been found reliable for early detection of liver damage or aplastic anaemia. Perhaps the most significant are a rise of the icterus index for the former condition, and a decrease of haemoglobin for the latter. Between the time of death and the first definite signs of symptoms of liver damage a median time of 34 days elapsed, and in the anaemia cases of 40 days. The treatment consisted in placing the patient on a high protein and carbohydrate diet, the administration of vitamin, liver, and iron preparations, infusion of glucose solutions, and blood transfusion. Treatment of acute hepatitis with methionine appeared hopeful. Post-mortem examination of the hepatitis cases showed a great reduction in the size of the liver. In some areas there was a complete destruction of all parenchymal cells. The cases of aplastic anaemia appeared to be independent of toxic jaundice: liver destruction was sometimes present, and also diffuse haemorrhages in all organs. *H. M. Vernon.*

Metabolites of 2, 4, 6–Trinitrotoluene (TNT) Excreted in the Urine of Dogs. SNYDER, R. K. (1946). *J. industr. Hyg.*, **28,** 59.

Munition workers are exposed to TNT in various ways, the more important being contact and inhalation of dust and fumes. Previous investigations indicated that skin contact or fumes were not capable of producing high urinary levels of TNT metabolites, though, in dogs, insufflation into the respiratory tract as well as oral doses did so. In 1921 H. H. Dale isolated 2,6–dinitro–4–azoxytoluene, and other researches followed. The methods used, which for the most part yielded doubtful or negative results, are too complicated to describe here. In the present tests oral doses of TNT at the rate of 50 mg. per kg. body weight were administered to dogs, who tolerated them well. By means of the *x*-ray powder diffraction pattern, the author was able to demonstrate that 2,6–dinitro–4–hydroxylaminotoluene was present in the urine of the dogs, and by means of extraction, separation, and the formation of an azo dye,

he was able to confirm Dale's rather doubtful identification of the presence of 2,6-dinitro-4-aminotoluene. Numerous tests, described in detail in the paper, failed to indicate the presence of TNT or of the five derivatives of it which were specially sought for. *H. M. Vernon.*

Endemic Fluorosis in Kweichow, China. LYTH, O. (1946). *Lancet*, **1**, 233.

All tribes and races in a wide area are equally affected. Investigations were done in two villages after a high incidence of dental mottling and spondylitis had been noticed in 1934. Spring water in the area contained from 2·4 to 13·1 p.p.m. of fluorine. Four cases are described, with pictures of two of them. The first is a woman of 60 with fluorotic enamel and complete fixation of the spine with considerable kyphosis; the second, a man of 58, had similar spondylitis and an enlarged cranium; the third case is of a man of 58 with mottled enamel spondylitis and limitation of arm and chest movements; the fourth is of a man of 37 with severe dental fluorosis and limitation of spinal movements. All have had pains of skeletal origin for years. A report on the exhumed skeleton of a man with severe spondylitis follows. The mandible was thickened and had exostoses, and the thyroid was thickened. The atlas was ankylosed to the occiput; the odontoid process was separated and ankylosed to the atlas; most of the lower vertebrae were fused together, and the spinal canal was greatly narrowed. The spinous processes were fused in a flat plate. The ribs and sacro-iliac joints were fixed. Most bones had exostoses, large ridges, and rough muscle attachments, and there were ossifications in the interosseous membranes. Death had occurred from fracture-dislocation between axis and atlas. Thickening of the skull was present here, but was not a constant finding. The teeth of 134 people, mainly children, were examined: 58 had moderate to severe fluorosis and 39 had mild changes. *J. N. Agate.*

Industrial Assessments: Some Problems and Suggestions. HEIM, A. W. (1946). *Occupat. Psychol.*, **20**, 24.

In this article assessment is synonymous with rating: both terms are used to connote a judgment of one person by another. The following problems are discussed. (1) *Halo effect.*—It is very difficult to get the assessor to concentrate on particular quality desired. For example, in answer to a question about her *efficiency at work*, Miss Brown may be described as 'very unsatisfactory' because she is frequently late arriving, but 'when she is here she is good—one of the best girls.' Halo effect includes the tendency (*a*) to base total judgment on some strong particular impression; (*b*) to let general impressions influence answers about particular quality. (2) *Range and distribution of assessment.*—All inexperienced assessors tend to rate judgment as about average. (3) *Absolute judgment.*—Assessors seem to have their own absolute judgment. (4) *Judgments on potentialities* rather than on actualities. (5) Tendency to take *other factors* into consideration. 'She's quite good at it, but then it's a very easy job.' (6) *Personal prejudice.* (7) *Lack of perspicacity.* (8) *Terminology.*—Assessors find difficulty in understanding meaning of terms.

The following suggestions are made. (1) Have more points on rating scale than are necessary. On a five-point scale inexperienced assessors tend to concentrate on two, or at the most three, grades, but the same assessors with a ten-point scale will use five grades. (2) Allow assessors scope for the passion for fine discrimination which is often found with unwillingness to make bigger judgments. Allow two assessments for each grade (for example, plus and minus). (3) Give brief description of what is meant by each grade of each quality. (4) Present lowest grade first in rating scheme. (5) Ask for notes on matters unrelated to particular set of inquiries: these illuminate characteristics of subject and assessor. (6) Obtain two independent assessments of each subject to compare consistency of assessors with each other and to encourage responsible rating. (7) All assessments should be signed. (8) In the case of new entrants, obtain assessments at three different times (for example, one, three, and six months after starting work). This checks consistency of assessments and gives some idea of the development of the subject. (9) Use paired comparisons, in which every subject is compared with every other subject. This can be done only for small groups (not more than a dozen), but it checks the consistency of the assessor (*a*) with other assessors, (*b*) with himself.

Conclusions are tentative as this investigation is incomplete. The number of statistically significant agreements between pairs of assessments is higher for total assessment than for particular qualities. The quality on which the least agreement obtains is 'ability to get on with management'—which is due to the assessor unjustifiably associating himself with the management. Paired comparisons, though possible for small groups only, and very time-consuming for the assessors, hold most promise for future developments.

T. A. Lloyd Davies.

How Does Housing Affect Health? POND, M. A. (1946). *Publ. Hlth. Reps.*, **61**, 665.

Though students suggest that those who live in good houses are healthier than those who live in sub-standard dwellings, proof of this is difficult. The morbidity and mortality rates for enteric infection and tuberculosis are significantly lower for families in good houses. Sharp differences in the respiratory rate and the rejection rate for draftees reflect the influence of bad housing. Common communicable diseases of childhood occur more frequently and at an earlier age in overcrowded households; the National Health Survey disclosed that the percentage of persons disabled for a week or longer was higher in households with more than one and a half persons to a room. The pneumonia rate was 68% higher. The National Health Survey disclosed that, of the houses visited, 4·0% had two or more persons, 6·3% more than one and a half persons, and 17·6% one person to a room. The mortality rate falls for infants born into families moved from slums to satisfactory housing. Slum areas in the District of Columbia showed rejection rates of Army draftees for personality disorders one and a half times higher than good housing areas. The total incidence of psychopathic and mental disorders is higher in the lower socio-economic group of the community. Sub-standard housing is not the only factor working to the detriment of health: the slum is a symptom of low economic status and is accompanied by overcrowding, illiteracy, poor nutrition, and delinquency. Good housing must provide fundamental physiological and psychological needs, and protection against contagion and accidents. Housing is not simply bricks and mortar: it is a complex structure of buildings and people. Careful management and the provision of adequate services are essential. Health authorities can assist in promoting healthy living (for example, diphtheria immunization), but they can take advantage of the community groups that spring up around housing projects, of selling the idea of public and personal health. Even in the U.S.A. there is an urgent need to devote individual and national effort to providing decent homes for millions who need them.

T. A. Lloyd Davies.

The Thymol-Barbital Test in Experimental Carbon Tetrachloride Poisoning. MCCORD, C. P., STERNER, J. H., KLINE, L. L., and WILLIAMS, P. E. (1946). *Occupat. Med.*, **1**, 160.

Maclagan, in an investigation on the colloidal gold reaction of serum, determined that thymol, introduced for antiseptic purposes into the barbital employed as a buffer, led to a turbidity when mixed with certain serums. This is specific for hepatic dysfunction and is attributed to the phenolic precipitation of a gamma globulin-phospho-lipin complex. Experimental observations were

made to establish the earliest evidence of organic damage in rabbits and rats from the inhalation of carbon tetrachloride vapour. Exposed to concentrations of 40, 60, and 80 parts per million, increase in thymol turbidity appeared after five days' exposure, with further increases after another five days. The thymol turbidity disappears by the end of seven days after the termination of exposure. The association of the more severe degrees of test reaction with well defined organic injury suggests that the responses at the above levels are indicative of incipient organic damage. *K. M. A. Perry.*

Poisoning by Barium Carbonate. MORTON, W. (1945). *Lancet*, **2**, 738.

Two attacks of food poisoning due to barium carbonate occurred in the Persia Iraq Command, 85 soldiers being involved. In the first outbreak 13 men were admitted to hospital. They were taken ill with acute gastro-enteritis an hour after their evening meal which consisted of meat, vegetables, and marmalade tart. Three sergeants refused the marmalade tart and were not affected, while five men who had not partaken of the meal, but had finished the marmalade tart, also developed gastro-enteritis. Chemical analysis showed there was no arsenic in the tart. Shortly afterwards 71 men from the same unit were taken ill two hours after a meal consisting of meat, vegetables, and treacle tart. A chemical analysis of the pastry revealed large quantities of barium carbonate. Investigation showed that at a supply depot a sack containing 4 lb. of barium carbonate for use as a rat poison had erroneously been put into the flour store, filled with flour, and issued to the unit. The symptoms consisted of a first phase of diarrhoea and vomiting with tingling sensations in the face and neck, followed by loss of tendon reflexes, irregular pulse, and muscle paralysis. In a few severe cases the paralysis became generalized and in one case led to almost complete respiratory paralysis. Recovery was rapid, and there were no deaths. *K. M. A. Perry.*

British Anti-Lewisite. PETERS, R. E., STOCKEN, L. A., and THOMPSON, R. H. S. (1945). *Nature*, **156**, 618.

A brief review, giving the main facts about the discovery of BAL. An attempt is made to focus the main stages of the discovery. Work by Peters (1936) suggested that the partial failure of an enzyme system, pyruvate oxidase, could be directly responsible for the pathological change brought about by vesicants. This, with other work, lead to the theory that a biochemical lesion in a tissue, formed by the partial blocking of an enzyme system, can cause pathological damage; the poison produces in effect an enzyme deficiency. The idea that the site of attack of trivalent arsenicals is an essential SH group in this enzyme-protein was confirmed. Thioarsenites of the type RAs $(SR^1)_2$ were shown to be dissociable in alkaline solution, so the toxicity might be reversible. Seventy-five per cent. of the arsenic in a lewisite-kerateine compound was found to be in combination with two thiol groups. Simple dithiol compounds might form relatively stable ring compounds with lewisite and thus compete successfully with 'dithiol' proteins in the tissues. The study of therapeutic possibilities began in 1940. After some trials the new compound 2·3 dimercaptopropanol was made and proved very satisfactory; this compound was named BAL by the Americans. Tests on the treatment of arsenical burns with BAL are described and experiments on the elimination of arsenic in rats during treatment, and on its toxicity. The use of BAL ointment in the treatment of arsenical accidents in factories proved very successful. The ointment was tried against the toxic complications of therapeutic arsenical drugs and then superseded by intramuscular injection. An ampoule, using as vehicle 10 per cent. benzyl benzoate in arachis oil, was prepared in the United States and very favourable results reported. In the cases tried in this country the results are much better than for any treatment so far tried. References are given to all the main stages of the work. *H. M. B. Buckell.*

Detecting Radiation Hazards. Current Comment. (1946). *J. Amer. med. Assoc.*, **130**, 791.

A recent release from the University of Rochester reveals many heretofore unpublicized aspects of research connected with the atomic bomb. The medical aspects of the research were organized in ten divisions. Among the most important was the development of methods to protect the employees; of the thousands employed in the plants not a single one was injured by any of the new and peculiar hazards of the work. Animal investigations on the toxicology and pharmacology of uranium compounds established criteria for tolerance levels. Methods of analysing the urine of employees were devised to determine the extent of exposure and to estimate the amount of radiation exposure which could be tolerated by human beings. A method of taking finger impressions in wax was devised and employed on the workers handling radio-active materials in atomic plants. This permitted the determination of when, if at all, employees began to show changes in the skin as a result of contact with the materials handled. One division studied the blood of large groups of animals, completing over 100,000 analyses. Geneticists studied the effects produced on the offspring of mice and fruit flies which had been exposed to radiation. Some 277,000 mice and 50 million fruit flies were bred and observed. The application of the vast amount of data gathered by the University of Rochester group, of which that cited is only an example, can hardly be predicted, but the results will certainly be significant in the fields of metabolism, cancer, haematology and physical medicine. *A. Thelwall Jones.*

Artificial Respiration. COMROE, J. H., Jr., and DRIPPS, R. D. (1946). *J. Amer. med. Assoc.*, **130**, 381.

The Schafer prone pressure and the Eve tilting technique of artificial respiration are the two most widely discussed methods at present, and the authors have now attempted to compare quantitatively their respective values in typical cases of respiratory failure with asphyxia. They were fortunate enough to find two patients with complete respiratory arrest, one suffering from a severe head injury and the other from an intraventricular rupture of a brain abscess, and both were unconscious throughout the tests. Respiration was measured by applying tightly a full face mask equipped with flutter valves, the expired air being conducted through a Bohr meter and measured. An endotracheal tube was used to ensure an open airway.

Results.

1. *Comparison of Schafer and Eve Methods.*—The authors showed quite conclusively the inadequacy of the Schafer method, in which the tidal air varied from 71·5 to 117 c.cm., whereas in the Eve technique the tidal air ranged from 286 to 500 c.cm. They point out that the Schafer technique, which depends for its action on the elastic recoil of the lungs and tonicity in the respiratory muscles, fails in patients who are unconscious, apnoeic, asphyctic and in shock, because such patients resemble a fresh cadaver more closely than they resemble a normal healthy subject and the elasticity in these subjects is reduced to a minimum. On the other hand, the Eve tilting method is efficient when tonicity and elasticity are absent.

As the time element in commencement of artificial respiration is most important, the Schafer pressure method must, obviously, be used as an emergency measure, but the authors insist that this method should only be used as a very temporary emergency measure, pending the commencement of the Eve tilting method. Further, they insist that the patient's care should pass from the hands of a 'first aider' at the earliest possible moment, to those of a physician highly trained in resuscitation.

2. *Intratracheal Insufflation with Oxygen.*—During their investigation the authors had an opportunity of observing the effect of insufflation with oxygen and were able to make observations on arterial and alveolar gas tensions during this procedure. The arterial oxygen saturation was 95·8 per cent. in one case and 90 per cent. in the other, but, at the same time the arterial carbon dioxide tensions were 166 and 142 mm. of mercury and the arterial *p*H values were 6·86 and 6·86 respectively.

Samples of alveolar air showed a concentration of 44 per cent. carbon dioxide, leaving no doubt regarding the accumulation of this gas, which may occur during respiratory arrest, despite intratracheal insufflation with oxygen. Thus, although the patients had a pink colour and were well oxygenated, the arterial blood analyses showed that the blood bathing the central nervous system was grossly abnormal in other respects. The enormous increase in carbon dioxide tension and the acidity of the arterial blood are factors not so readily appreciated by the clinician as is a decrease in arterial oxygen saturation. The arterial *p*H values in these two patients are among the lowest ever recorded, similar figures having been reported only in terminal nephritis, and this acidity, together with the high concentrations of carbon dioxide present in the blood, undoubtedly exerted a narcotic depressant effect. Hyperventilation with an anaesthesia bag at the rate of 40 cycles per minute in one patient reduced the arterial carbon dioxide tension to 106 mm. of mercury and increased the *p*H to 7·00.

These studies emphasize that artificial respiration, to give maximum efficiency, must not only raise blood oxygen to normal levels, but must also reduce blood carbon dioxide to normal levels, and this is impossible unless intratracheal insufflation with oxygen is combined with rhythmic compression of the chest.

The authors have produced quantitative data which cast grave doubt on the efficacy of the Schafer prone pressure method for producing artificial respiration, but so few cases are available, up to date, that it is of the utmost importance that clinicians and physiologists should collect data on many more apnoeic, asphyxiated patients in whom need exists for artificial ventilation.

A. Lloyd Potter.

Contact Dermatitis. An Analysis or Tabulation of All Cases Proved in a Single Year. HOWELL, J. B. (1946). *Arch. Dermat. Syph., Chicago*, **53**, 265.

The importance of contact irritants employed as topical medications or encountered as occupational hazards is discussed. All 250 cases were proved by patch tests.

1. *Dermatitis from topical applications.*—This was the commonest cause of contact dermatitis, sulphonamides and mercurials being the most potent sensitizers. Warning is given of the danger that patch tests with sulphonamides may photosensitize the patient or cause severe aggravation of the existing eruption; small test doses given orally are preferred to confirm sensitivity.

2. *Contact dermatitis of occupational origin.*—Of a total of 86 cases, 23% occurred in the aircraft industry, from formaldehyde and phenol formaldehyde (for plastics) glue, and rubber gloves; zinc chromate primer was the commonest sensitizer.

Attention is drawn to the importance of oleoresins in weeds as an occupational hazard to farmers and outdoor workers—35 (41%) of the total occupational cases being so caused. Weed dermatitis usually begins in the spring on the ankles, and later affects other exposed parts; it recedes in winter, but recurs more severely each succeeding spring. Polyvalent sensitization is the rule. Two forms of management of such dermatitis are advocated: (*a*) absolute avoidance of vegetation; (*b*) oral administration of specific weed oils during winter months.

Nickel is often the sensitizing agent in cases from jewellery or wearing apparel; and, in the list of 58 cosmetics implicated, 20 occurred from nail polish and 21 from hair lacquer. *Geoffrey A. Hodgson.*

Protection against X Rays and Gamma Rays. X-Ray Protection in Industrial Fluoroscopy. HENNY, G. E. (1946). *Radiology*, **46**, 66.

This article is part of a combined report for the Standardization Committee of the American Röntgen Ray Society and Radiological Society of North America. The object of the report is to acquaint those who have recently become engaged in the use of *x* rays and radium with the actual hazards involved, and also to reassure them against imaginary and mythical dangers. Protection in industrial fluoroscopy differs from that in radiography because higher voltages are used and because the tube operates for longer periods in fluoroscopy, making the total *x*-ray output greater. The operator and other workers in the room must be protected from the direct beam of *x* rays and also from scattered radiation. The thickness of the protective material must be adequate for the kilovoltage used, and the whole apparatus must be tested for leakage of radiation. Special warning of the risk of the operator's hand entering the direct beam of rays when adjusting or moving the specimen is given, and the need for some safety device to prevent this is stressed. Care must be taken to prevent leakage around the ends of the long castings which may project from the fluoroscopic housing and from the ends of the enclosure when specimens are brought to the fluoroscope on conveyor belts. In all cases it is possible to install adequate protection by efficient planning. The method of detecting leaks and checking the efficiency of the protection are discussed. The Geiger-Müller counter is recommended. The Counter and the way in which leaks may be detected, and the adequacy of the protection tested are described. Other methods are mentioned. Two monthly blood counts of all workers exposed to *x* rays are advocated.

M. H. Jupe.

Pulmonary Changes in Carbon Tetrachloride Poisoning. THOMPSON, C. M. (1946). *Amer. J. Röntgen.*, **55**, 16.

Of 20 men poisoned by inhalation of carbon tetrachloride fumes, four showed evidence of changes in chest radiographs. In one case, which proved fatal, almost the whole of the lung fields was homogeneously dense, due to what was found at autopsy to be congestion and consolidation with acute pneumonia—haemorrhagic in type. In the other three men, examined seven days after exposure, there was increase of hilar shadows and prominence of lung markings. The signs had disappeared by the tenth day. All four of the patients showed evidence of toxic renal syndrome—swelling of soft tissues, oliguria, albuminuria, vomiting, etc. The carbon tetrachloride was being used for cleaning purposes and was not subjected to any heat which might alter it. The post-mortem findings are described. *M. H. Jupe.*

Pneumoconiosis Due to Cotton Dust (Byssinosis) MCCARTHY, P. V., and AKENHEAD, W. R. (1946). *Radiology*, **46**, 46.

A short account of the features of byssinosis and of the work of Prausnitz and Fletcher for the Medical Research Council are given. The history of a case and description of the radiological findings follow. The diagnosis is based on the history of employment and on the medical history, together with the fact that the radiographic picture did not change in two months. The radiograph included in the article is not that usually met with in byssinosis. *M. H. Jupe.*

Symposium on Scientific Proof and the Relations of Law and Medicine—Effects of Electrical Injuries, with Particular References to the Nervous System. HYSLOP, G. H. (1946). *Occupat. Med.*, **1**, 199.

In 1941, in the United States, there were 388 deaths due to lightning and 890 deaths due to other electrical currents excluding judicial electrocution. Reliable and comprehensive records about non-fatal electrical accidents are virtually non-existent, but it is deduced that

about 17% of electrical accidents are fatal. About 0·4% of all accidents in industry and in the home are due to electricity. The resistance of the human skin in calloused parts may reach 1,000,000 ohms, but the average dry skin is about 5,000 ohms, and the moist skin 1,000 ohms or less. In a given period the effective voltage of an alternating current is 0·707 that of a direct current of the same voltage, but at certain phases of the alternating cycles the maximum effective current is 1·41 times that of the direct current per volt.

In the United States the commonest voltage for domestic purposes is 120; industrial current commonly has a voltage of 120–240, and electrical railways 600. Overhead transmission may be up to 30,000 volts. Currents with frequency cycles of over 2,000 become decreasingly dangerous.

There is no experimental evidence to support the idea of many workmen that they are able to acquire resistance to electricity. Such claims are dangerous and based on tricks in the manner in which they make contact. When electricity enters the body its effects depend on the tissues traversed and the strength of the current. Low currents produce a physiological effect, such as contraction of muscles. The function of the living tissue is accompanied by electrical activity, measured in micro-amperes, which may be made of diagnostic use in such instruments as electrocardiograph, electromyograph, and electroencephalograph. The direct effect, either physiological or pathological, of electricity passing through the body is limited to the tissues traversed by the current, and the current always takes the shortest route through the body. At a given point in the path all tissues conduct the same amount of current, and in the tissues adjacent to, but away from, the main path of the current, the effect decreases rapidly. In animals, if the shock passed from forefoot to forefoot, forefoot to hindfoot, or hindfoot to hindfoot, no current could be detected in any part of the brain, but when the current passed from forefoot to forefoot a significant current could be detected in the spinal cord.

The critical level for producing lasting disturbances in nerve tissue is 30 milliamperes per 3 mm. nerve diameter for shocks of 5 seconds' duration. Currents of 40 milliamperes produce irreversible damage. Current passing through the brain damages the cells of the cortex more than deeper tissues. Prolonged convulsions, protracted coma, and prolonged arrested breathing follow, and if the current passes through the respiratory centres there will be depression or paralysis of respiration. If the current passes through the heart the rhythm will be disturbed. Death caused by electrical current, either immediate or in a few hours, results from (1) ventricular fibrillation, (2) failure of respiratory centre, (3) a combination of the above, (4) prolonged tetanus of respiratory muscles.

In non-fatal shocks, damage to the brain may be either localized or diffuse, and results in epilepsy, hemiplegia, etc. After an electric shock pre-existent organic disease of which the victim is not aware may be made worse or noticed for the first time.

Claims of organic damage to the brain caused by electric currents should be regarded with scepticism unless there is evidence of adequate exposure having occurred. The cranial nerves are seldom affected. If there has been immediate and severe organic damage to the brain psychosis may follow, but more likely constitutional psychoses such as dementia praecox or manic-depressive insanity are aggravated. Psychoneurotic symptoms such as anxiety, phobias, and obssesions are common. Malingerers can generally be exposed by considering the circumstances of the accident and their subsequent behaviour.

In the treatment of electric shock it is usually impossible to distinguish between respiratory failure and ventricular fibrillation. Action should be based on the assumption that unconsciousness is due to respiratory failure. Immediate and persistent artificial respiration is important; of 361 victims treated within three minutes of the accident, 70% recovered. If there is clinical evidence that the heart is not functioning, the drastic measures of direct injection to the heart or cardiac massage have been suggested. *T. A. Lloyd Davies.*

Medico-legal Aspects of Injuries from Exposure to Röntgen Rays and Radioactive Substances. DUNLOP, C. E. (1946). *Occupat. Med.*, **1**, 237.

High voltages from 100,000 to 1,000,000 volts are used for generating rays for therapeutic purposes. Approximately 600 'r' generated at 200,000 volts produce moderate erythema of the skin. Hazards due to radiation arise in the mining of radio-active ores; the manufacture of *x*-ray tubes, metal casing, luminous dials, radium applicator needles, the examination of metal casing, biological, chemical, and physical experiments with radiation, and the use of *x*-ray apparatus by untrained persons, or in the promotion of sales, for example, shoe-fitting machines.

Since *x*-ray machines used for diagnostic purposes are worked at low voltage they produce a soft radiation of low penetrating power. Injuries are, therefore, almost exclusively confined to burns of the skin. A high proportion of all accidental burns are incurred during fluoroscopy where attempts are made to set fractures or remove foreign bodies in direct fluoroscopic vision.

The term 'radiation burn' is usually reserved for tissue reactions of undesirable severity, though the layman may apply the term 'burn' to reactions of the epidermis produced in routine tumour therapy. Such a reaction heals, but the skin is subsequently atrophic and pigmented.

The physician is much more likely to be criticized for the results of over-treatment than for under-treatment. Inadequate treatment may result in the inability to arrest malignant disease, and in the death of the patient: at the least it delays the institution of effective therapy or sets up a state of radio-resistance.

Acne vulgaris often responds well to *x* rays, but as the condition responds to conservative treatment the physician is strictly accountable for over-zealous or careless radiation. Malignant melanoma is, on the other hand, fatal, regardless of the type of treatment, and the physician must consider whether the meagre hope of treatment justifies the danger and discomfort of full irradiation. A more lenient attitude of the courts towards burns incurred in the course of legitimate and proper treatment is needed.

Thorium is radio-active and is sometimes used diagnostically. The Council of Pharmacy and Chemistry of the American Medical Association, reviewing the possible dangers of injecting thorotrast, state that 'most investigators advise that the material (thorium dioxide) be employed only in cases in which there is some serious reason for using it or in those in which early death may reasonably be expected.'

Radiologists can usually avoid exposing themselves to a beam of direct radiation, but they are endangered by the incidental radiation present in the vicinity of *x*-ray tubes and radium. They are occasionally unwise enough to place their hands in the beam of radiation: the dose may be small, but the effects are cumulative. Burns of the hands and fingers result from manipulating radium needles with the fingers instead of with long forceps. Dentists should ask the patient to hold a dental film in position during exposure. Scattered radiation is seldom sufficient to produce localized damage of the skin, but leads to fatigue, headache, anorexia, and to damage of radio-sensitive tissues such as ovaries, testes, and blood-forming tissues. The daily tolerance dose is 0·2 r. Routine medical examination of persons exposed to radiation should be carried out four times a year, and a blood count once a month. Protection is grossly inadequate in many hospitals. The chief defences of radiological workers against over-exposure are distance, speed, and lead. Lead should be used liberally to reduce scattered radiation to a minimum.

There is no danger in handling tissues or objects after

irradiation: no toxic radiation or poison is 'stored' in them. Any living cells can be destroyed if subject to sufficient radiation, but some are more sensitive than others. The most sensitive tissues are lymphocytes, bone marrow cells (myeloblasts, erythroblasts, megakaryocytes), and germ cells (testes and ovaries), and the least, cartilage, muscle, and brain and nervous tissues. Immature and undifferentiated cells are more sensitive than mature cells, which accounts for the increased radiosensitivity of tissues of children. Different functions of a cell can be depressed in different degrees, and cellular death does not necessarily follow irradiation. The most easily damaged function is the ability to reproduce by mitosis. Stimulation of cells or organs by small doses of radiation has never been demonstrated. Exposure to even a large dose of radiation produces no sensation: the period between exposure and reaction is the latent period; after irradiation, permanent change may occur in tissues in the absence of demonstrable alteration: for example, skin exposed to a dose sufficient to produce erythema is more vulnerable to subsequent irradiation. The dose which different persons can tolerate cannot be forecast with accuracy. Most of the damage resulting from radiation occurs in the path of the beam and is localized to the skin: general effects are radiation sickness and blood changes.

Comparatively little is known about the nature of tissue reaction to radiation: tissues retain their normal appearance, and only after a lapse of 10–14 days is erythema of the skin, which marks the clinical onset of reaction, to be expected. In the skin erythema, oedema, and inflammation is followed by loss of elastic tissues in the walls of arterioles and connective tissues, and proliferation of endothelial cells of arterioles and capillaries; the lumen is blocked, and, as this reaction subsides, pigmentation, fibrosis, and atrophy of the dermis supervene. Cancer frequently arises (at periods from half to thirty years) in radiation burns: of 135 physicians seeking advice for skin burns due to irradiation between 1919 and 1939, 29% had cancer at the site of the burn.

Growing bones of infants and children are easily damaged (by 25% and 50% of the dose producing erythema in adults). The damage is manifest by impaired growth. Heavy irradiation of adult bones may result in spontaneous fracture, osteitis, and necrosis; and, in the jaw, if the gums or teeth sockets are infected, infection and destruction of the bone is a serious risk. Radioactive substances stored in the bone may cause bone sarcoma: many of the disabilities ascribed to radiation osteitis are due to infection. Mature cartilage damaged by radiation shows little tendency to repair. The gastro-intestinal mucosa are moderately sensitive to *x* rays and radiation; a temporary gastritis is common, and infection, ulceration, and obstruction of the sigmoid colon are stated to be frequent. A change in the number and character of the blood cells is characteristic of radio-activity, but radiotherapy as usually conducted carries little risk of aplastic anaemia. The germicidal epithelium of the ovaries and testes is easily damaged and destroyed. Sterilization of men and women may be effected by *x* rays: in women this is usually concomitant with the treatment of pelvic malignant disease—though it may be used as a method of birth control. Children conceived after irradiation of the ovaries or testes show no defects or damage (in contradistinction to animal experiments). Married female radiologists show an incidence of 24% of sterility, but no increase in abortions or abnormal children. Children irradiated *in utero* may be killed or show congenital defects: the commonest defect is microcephaly. A pregnant woman may recover damages for personal injuries, but not for a child killed *in utero* by irradiation. A husband has no right of action to recover damages for a child destroyed *en ventre sa mère*.

T. A. Lloyd Davies.

Tin Mining and Silicosis in Cornwall. HALE, L. W. (1946). *Thorax*, **1**, 71.

In Cornwall the metalliferous lodes occur in fissures in the granite and lie roughly in the vertical plane. The metals occur at varying depths in the fissure, lead and zinc being the most superficial, copper next, and tin deepest. The tin occurs in the form of stannic oxide, and the extraction of this from the lode rock is briefly described. A ton of good ore may yield 25 lb. stannic oxide, of which 75% is metal. All drilling is done with pneumatically-operated axial water-feed percussion drills, the bit striking the rock some 2,000 blows per minute and rotating on its long axis about 120 times per minute. Construction and types of drill are indicated. Environmental facts mentioned include: that the granite has a total silica content of about 80%; that, as this is a compact rock, falls of the roof are comparatively rare; that owing to naturally occurring water and the use of sprays, etc., for dust-control the workings are wet; that gas does not occur in tin-mines and naked-flame lamps are used; and that wet-bulb temperatures in the stope-cavities range up to 90° F. or higher. Mines at present working are up to 2,000 feet deep.

Dust-risk is greatest in the operations of drilling, shot-firing, and tram-loading. Dust-counts are not yet available, but it is stated that visible dust is rare during a working shift. Dust-prevention measures in use include: (1) 'natural ventilation,' in which convection currents are utilized to distribute air from the surface through the workings: no fan-ventilation is used; (2) wet-drilling, which has been the greatest single influence in dust-control in this industry; (3) mist-projectors, which spray atomized water or a mist of oil and water during and after shot-firing; (4) hand-operated sprays for watering down the piles of broken ore before tram-loading; (5) a time-schedule whereby shot-firing is done only at the end of a shift, after which the mine is left empty for not less than three hours, during which time the workings are blown through with compressed air; and (6) the use of compressed-air rings to ventilate blind-end workings, etc. Some difficulties in dust-prevention are mentioned, including that of ensuring proper application of the regulations, and it is suggested that systematic education of the miner in this respect should be undertaken.

The macroscopic and microscopic appearances in tin-miners' silicosis are described. It is stated that the average exposure before nodulation occurs is 15 years. The symptoms are not invariably proportional to the radiological findings; they include dyspnoea (often of asthmatic type in advanced cases) and cough, and are accompanied by the physical signs of emphysema. In the absence of major infective complications the presence of nodular fibrosis may be compatible with a full life expectation, and it may be that the nature and degree of secondary infection in the lung is a decisive prognostic factor. At necropsy the majority of cases are found to have pulmonary tuberculosis, other causes of death being non-tuberculous pulmonary infective states, and congestive heart-failure. The radiological appearances of tin-miners' silicosis are described and compared with those of coal-miners' pneumoconiosis; it is suggested that a standard might be agreed for the degree of radiographic shadowing necessary to constitute 'reticulation.' The problem of disability-assessment by some method of measurement (rather than judgement) is discussed, and it is stated that respiratory disability may vary considerably from week to week (with weather-conditions, etc.) in the silicotic miner.

L. W. Hale.

An Outbreak of Scabies in Stenographers. MARKEL, J. (1946). *Occupat. Med.*, **1**, 386.

Fifteen women working adjacent to each other in a group of 50 suffered from scabies transmitted from the original case by passing papers from one to another.

T. A. Lloyd Davies.

PROCEEDINGS OF THE ASSOCIATION OF INDUSTRIAL MEDICAL OFFICERS

FORTY-THIRD MEETING

The forty-third meeting of the Association was held on Friday, March 22, 1946, at the London School of Hygiene and Tropical Medicine. Dr. W. Blood was in the chair.

Circulation of Minutes

The Hon. Secretary reported that the Executive Committee were not in favour of circulating the minutes of each meeting to members, as the expense would be heavy and as a summary was published in the *British Journal of Industrial Medicine*.

Nursing Committee

Dr. Catherine Swanston presented the report of this Committee for the past year. The Committee had been mainly pre-occupied with the training of the industrial nurse, and was hoping to prepare a report on the subject. The following were elected to the Nursing Committee for 1946: Dr. A. J. Amor, Dr. J. G. Billington, Dr. E. H. Capel, Prof. R. E. Lane, Dr. N. G. Marr, Dr. P. Pringle, Dr. Patricia Shaw, Dr. Catherine Swanston.

Foreign Relations Committee

Dr. Donald C. Norris, chairman of the Committee, presented the report for 1945. Contact had been made with the Offices of the Dominions, so that industrial medical officers visiting this country may be invited to meetings of the Association or its Groups. He asked members to let the Hon. Secretary know of any names of people prominent in the field of industrial medicine residing abroad, so that a register could be kept for the use of members proceeding overseas. The possibility of holding an International Congress of Industrial Medicine in this country was being explored. The following members were elected for the current year: Dr. A. J. Amor, Dr. J. C. Bridge, Dr. Elizabeth Bunbury, Dr. W. E. Chiesman, Dr. A. D. Forgie, Dr. Donald Hunter, Dr. G. F. Keatinge, Prof. R. E. Lane, Dr. T. Gwynne Maitland, Dr. E. R. A. Merewether, Dr. M. W. Goldblatt, Dr. Sibyl Horner, Dr. Donald Norris, Dr. P. Pringle, Dr. R. S. F. Schilling.

B.M.A. Standing Committee on Industrial Medicine

The Hon. Secretary was instructed to send a letter, approved by the meeting, to the Secretary of the B.M.A. requesting that that Association set up a Standing Committee in order to deal with the important problems arising in industrial medicine at the present time.

B.M.A. Industrial Medical Services Sub-Committee

The following members were appointed to represent the Association on this Committee: Dr. W. Blood, Dr. C. E. Graves Peirce, Dr. M. W. Goldblatt, Dr. P. Pringle.

Public Business

Dr. H. F. Chard read a paper on the practical treatment of minor industrial injuries. He distinguished between the major type of injury, which involved giving up work, and the much more common minor type, which could be efficiently dealt with at the factory. The degree to which the industrial medical officer personally undertook treatment depended to a great extent on the compactness of the works under his care. Where these were scattered, he had to rely on his nursing staff, for whom he emphatically advised the laying down of a definite procedure in the matter of treatment. After thorough cleansing of a wound, the dressing should be left undisturbed as far as possible. If a stitch were required—and he preferred stitching to clips or other mechanical device—a little care and thought in the sewing was well repaid in the final result. A plaster slab should be used if there was likely to be tension on the sutures from movements at work. After further review of minor fractures, removal of foreign bodies, tenosynovitis, and foot injuries, he concluded with a brief summary of his own experience of penicillin when used in a works ambulance room.

Dr. F. H. C. Beards followed with a paper on a system for the recording of sickness absence in industry with the object of determining occupational factors of causation. He pointed out that, whilst considerable efforts were made in industry to prevent accidents and to reduce time lost from this cause, yet, in the case of sickness, occupational causes of absence had not yet been satisfactorily differentiated from non-occupational. On the one hand, diseases such as silicosis could not be other than occupational, and on the other it was difficult to assign an environmental aetiology to uterine fibroids. Between these two extremes lay an uncharted area. It was important that more research should be undertaken, since non-occupational causes of sickness should be approached by the general health services of the country, and occupational causes should be tackled by industry itself. The two requisites for dealing with this problem were, first, an adequate system of records, and, secondly, a method of extracting information from them. The basis of the recording system must be the individual sickness absence record so designed that a machine could sort out any possible combinations of the records which might be required. The actual cause of absence should be based on the certificate of the private doctor, but in the case of absences of less than three days the employee's own statement should be relied on. Dr. Beards proceeded to describe in detail the system of investigation which had recently been set up in his own factory. It was too early to assess the results, but he hoped that on a long-term programme an analysis of the figures would indicate groups of workers with an abnormal severity rate of sickness absence which would give grounds for closer investigation into the cause.

By kind permission of Dr. W. S. C. Copeman, who welcomed the Association, the meeting was continued on Saturday, March 23, 1946, at the Rheumatism department of the West London Hospital. Dr. O. Savage spoke on osteo-arthritis in industry. He was followed by Dr. D. Wilson, who described epidemic myalgia, and by Dr. D. Prieskel, who spoke on the social and industrial background to rheumatism. Dr. A. C. Elkin gave a demonstration of treatment of early cases of rheumatic disease. The staff of the hospital showed members modern methods of physical medicine; and in conclusion Dr. W. S. C. Copeman gave a lecture on recent theories of the causation of fibrositis.

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